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BRAIN:

A JOURNAL OF NEUROLOGY.

EDITED BY

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ERRATA.

The Abstract of Déjérine's paper on "Nervo-Tabes" has accidentally escaped revision, and contains a few inaccuracies, which, however, do not alter the meaning.

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BRAIN.

APRIL, 1883.

Original Articles.

ON THE PUPIL IN EMOTIONAL STATES.

BY SAMUEL WILKS, F.R.S.

THERE is probably no structure of the body so mobile or so susceptible of great changes as the iris. The pupil of the eye, by the size of which these changes are perceived, is ever enlarging or diminishing under influences of the most varied kinds. These pertain in health to the impingement of light on the retina, and to the accommodation of the eyes to focal distances; in disease to numberless affections of the brain, spinal cord, and sympathetic system, and even to the more subtle changes which take place in the nervous system from the circulation of poisons in the blood. The pupils, too, may often be observed to react to stimuli applied to the face and neck. All these numberless causes acting on the eye are well known, but I now wish to draw attention to the changes of the iris under purely mental or emotional states.

I have on several occasions whilst sitting at the bedside of patients observed a quietude come over them in which they have assumed a fixed gaze; at the same time the pupils of the eyes have become dilated, and on the patient being suddenly aroused have as quickly contracted. These patients have fallen into the state known as hypnotism, which is identical apparently with that of somnambulism. The latter is not true sleep, where all the senses are for the time dead, for the somnambulist can walk in dangerous places without falling, showing that the eyes are still impressionable and capable of guiding the body. When therefore Lady Macbeth rises from her bed, walks out with a taper in her hand, and the doctor says, "She is fast asleep although her eyes are open," the gentlewoman is wrong in exclaiming "Ay, but their sense is shut."

I think there is every reason to believe that when the mind is in a passive state, or in any grade of that condition which might culminate in hypnotism or trance, the pupil dilates and the eyes look into space; thus in any pensive, dreamy or pleasurable mood the pupil dilates. When Poe exclaimed in poetical phrenzy:—

> "I cannot write—I cannot speak or think—
> Alas! I cannot feel, for 'tis not feeling,
> This standing motionless upon the golden
> Threshold of the wide-open gate of dreams,
> Gazing entranced adown the gorgeous vista,"

the poet might be pictured as literally standing gazing with "wide-open pupil."

In mental activity, on the contrary, in concentration of the mind on any object, or in the violent passions of terror or rage, the eye would be fixed on something near, and the pupils would be contracted. Whether there be such a fixed law in the lower animals I cannot say, but in my parrot it certainly obtains. The iris of the bird is ever oscillating, and its variations correspond to its different moods. When frightened, and most markedly when angry, the pupil contracts; when in an amiable frame of mind, the pupil widely dilates. The pupil is an accurate gauge of its mental state.

Poets and painters have in all times regarded the eye as an exponent of the state of mind, or rather of the passions of the soul, but they have never (so far as I know) with one exception attempted to analyse the reasons for this. Shakspeare in numberless passages speaks of the various expressions of the eye, as, for example, in those lines which are ready at hand, "the eye discourses, I will answer it," in 'Romeo and Juliet,' or "an eye like Mars to threaten and command," in 'Hamlet.' Tennyson speaks of good, kind, and pure eyes. Byron was always eloquent over woman's eyes, as in the stanza on the eve of the battle of Waterloo: "Soft eyes looked love to eyes which spoke again." The only allusion to the form of the eye as indicative of expression is the recognition of a wide or large eye in connection with modesty, affection, and simple love, in distinction from the small, hollow or retracted eye. As just now said, when the eye accommodates itself for the vision of near objects the pupils contract, whilst in looking at a distance the pupils dilate. Now in these different circumstances the mental state is different; in the one case the mind is engaged on some concrete object immediately in view, whilst in gazing into space the mind is in a more abstract mood. It may be that it is this want of near concentration of the eyes, and the consequent distant gaze which he assumes, which contributes to our idea of the majesty of the lion. This is well portrayed in Landseer's lions in Trafalgar Square. I might suggest that if the origin of the word "belladonna" is due to the plant producing an expansion of the iris, it is obvious that a dilated pupil is considered a sign of beauty, and this assuredly would be associated with a kindly and sympathetic state of the heart rather than with an active and powerful intellect.

Lavater speaks much of the expression of the eye, but attempts no analysis. He quotes Winkelmann to show that the eye is made to vary much in shape and size in the antique statues, and he also quotes Buffon in the following passage. "The images of our secret agitations are particularly painted in the eyes. The eye pertains more to the soul than any other organ; seems affected by and to participate in all its emotions; expresses sensations the most lively, passions the most tumultuous, feelings the most delightful, and sentiments the most delicate. It explains them in all their force, in all their purity, as they take birth; and transmits them by traits so rapid as to infuse into other minds the fire, the activity, the very image with which they themselves are inspired. The eye at once receives and reflects the intelligence of thought and the warmth of sensibility; it is the sense of the mind, the tongue of the understanding." If this be true, the eye stands pre-eminently above all the other organs of sense; it is not merely a recipient to carry to the brain images of the outward world, but it is again the exponent of the operations of the mind within. May not this afford a clue to the meaning of its complex mechanism? If, as Buffon says, it is the "sense of the mind," it is surely something more than a camera obscura.

The only author with whom I am acquainted who has actually remarked the condition of the pupil in emotions of the mind is Balzac, and his observations accord with what I have previously enumerated, and with the anatomical facts observed in my parrot. My knowledge of the writings of Balzac is but limited, but I heartily agree with the common judgment that few authors show so great an insight into character, and especially physiognomy, as this great novelist. I will quote from a book at hand, "Le Curé du Village." In this novel he describes his heroine as devoting herself to the Church and works of piety; she was always early at her prayers, and when before the altar her whole visage was transformed. "Véronique was for some moments changed. The pupil of her eyes, endued with a great contractility, appeared then to expand and draw back the blue of the iris until it formed no more than a narrow circle. Then the metamorphosis of the eye, active and lively as that of the eagle, completed the strange alteration of her countenance. Was it a storm of contained passion? Was it a force arising in the depths of the soul, which so enlarged the pupil in full daylight (as it grows large in ordinary life in the shade) and obscured the azure of those celestial eyes?" Véronique was, however, open to human impressions, and her heart was not proof against the attractions of a Government official who came to the village, although she refused his offer of marriage. On leaving her "he kissed her hand with a lively expression of regret, and the bishop remarked the strange movement by which the black of the pupil encroached on the blue of Véronique's eyes, which was reduced to only a slender circle. The eye evidently announced a violent revolution within."

On making enquiries amongst friends, in order to ascertain the accuracy of these statements, I have found that some persons have, without any consideration of the subject, made similar observations for themselves. For example, I have been told that if a gentleman has entered a room where a lady has been present who has had a *penchant* for him, her eyes have lighted up and her pupils dilated. The lighting up or brightening of the eye is probably due to increased vascularity, and participates in the general suffusion or blushing of the upper part of the body. This is shown by the brightness of the eye in pneumonia and bronchitis.

Balzac speaks of Véronique at church resembling the pictures of the Holy Virgin or adoring saints, and implies that they have the same expression of face which he describes. It would be interesting to know, therefore, in what way the eyes were delineated to produce the ecstatic expression of which he speaks. I have not been able to learn what these early Italian religious painters exactly portrayed, but it is very probable that they, as observant men, were able to produce an expression whose details or meaning they had never analysed. In looking at Rossetti's pictures, now being exhibited, I believe I can see a difference in the way he has portrayed the eyes. The artist must have wished to put into his faces different expressions, or wherefore does the catalogue speak of the pitying eye, &c.? Of course expression is given by prominence of the eye, its direction, and position of the lid (during life by movement), but I think I can perceive in such a picture as the "Blessed Damozel" (the larger one), the "Roman Widow," and more especially in "A Vision of Fiametta," a large full eye, distantly gazing, accompanied by a pupil of good size—

"The blessed damozel looked out
From the gold bar of heaven;
Her eyes were deeper than the depth
Of waters stilled at even."

I refrain from further remarks, lest it might seem that I am writing at the dictates of my fancy, and forgetting that my object is to say no more than the limits of a scientific journal allow. I will therefore leave with the reader the simple statement that, as in somnambulism, ecstasy, catalepsy and allied conditions, the pupil is widely dilated, so is it, although in a less degree, in such mental moods where all the senses are hushed, and serenity, benevolence and the pure passion of love prevail. If this be true, it shows that the eye is not only a passive organ and one of the gateways of knowledge, but is also a portal through which the working of the brain becomes manifest. I have little doubt that a good deal of physiological knowledge on this interesting subject exists amongst good observers in an unwritten form, and I therefore trust that this short paper may be the means of eliciting some facts from others, in illustration of the Scripture truth, that "the light of the body is the eye."

ON CHRONIC ATROPHIC SPINAL PARALYSIS IN THE CHILD; AND ON A RARE MODIFICATION OF THE REACTION OF DEGENERATION.

BY W. ERB, M.D.,

Professor of Medicine to the University of Heidelberg.

It is not very long since clinical, experimental, and electro-diagnostic researches have made it possible to define and establish the morbid type called "Atrophic paralysis." We understand thereby all such motor paralysis with which is associated a rapid disappearance of the muscles, due to the degenerative atrophy of the motor nerves and of the muscular tissue. The presence of this atrophy is demonstrable by an electrical investigation of the tissues which then present the reactions of degeneration[1] more or less fully developed. Notwithstanding the general uniformity of the picture presented by atrophic paralysis, a closer investigation has shown that it may arise from morbid processes of different nature and variable localisation. Pure cerebral paralysis, however, arising from lesions situated above the pons, are never accompanied with degenerative atrophy and R.D.—(except in the case of certain rare complications and of secondary changes). Degenerative reactions, on the other hand, are frequently observed in numerous kinds of spinal and peripheral lesions, under definite circumstances. Hence, atrophic peripheral paralyses have with right been brought by the side of spinal paralyses.

Every possible lesion of the peripheral motor nerves—provided they be deep enough to create a complete break of conductivities—may lead to atrophic paralysis. Thus the so-called "rheumatic" affection of certain nerves (e.g. of the facial), but much more frequently the mechanical and traumatic lesions of a nerve (compression, crushing, dividing, tearing), and finally a sufficiently developed neuritis, lead to a complete destruction of the conducting paths in the nerve. The last-mentioned cause, neuritis, has lately been made to assume a much too prominent position; though it certainly occurs in connection with atrophic paralysis both isolated in single trunks, or diffused as "multiple neuritis," yet its occurrence is not so frequent as some authors believe. It is by no means proved that all which now goes under the name of neuritis is so in reality; post-mortem appearances have often displayed the closest analogy with the degenerative atrophy of the nerve (of neurotic origin), and cannot without further proof be admitted as "neuritis." It is conceivable that this degenerative atrophy is set up in the nerves themselves (e.g. by certain poisons, lead, ergotine), but it is perfectly admissible also that it depends upon a functional disturbance in the anterior spinal cornua, the nature of which as yet escapes our means of microscopical investigation. Scheube[1] and Bälz,[2] basing themselves upon good observations and sound reasoning, have advanced the view that the Japanese *Kak-ke*—that peculiar endemic disease which occurs so frequently in Eastern Asia, as well as in other countries (under the name of *Beri-beri*)—is nothing but an infectious inflammation of the peripheral nerves; should this theory be confirmed, multiple neuritis would acquire a meaning of considerable importance. As far as Europe is concerned, however, this fact would perhaps only serve to stimulate thought and research with reference to the few scattered cases of multiple neuritis observed here.

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[1] [The abbreviation R.D. may conveniently be adopted to stand for "Reaction of Degeneration"—a clumsy expression for which, however, it is difficult to find a substitute at once short and correct.—A. DE W.]

The spinal atrophic paralyses are doubtlessly to be referred, in almost every instance, to disease of the anterior grey matter of the cord—the possibility being excepted, in some rare instances, of a lesion of the anterior roots within the cord, before they reach the grey matter. It is still doubtful whether degenerative atrophy of spinal origin implies always destructive lesions, recognisable microscopically by the destruction or degeneration of the large multipolar ganglionic cells. It is, as just mentioned, quite possible that functional, microscopically unrecognisable disturbances of these cells (e.g. toxic substances) may have the same effect. This effect would obviously manifest itself by symptoms of paralysis and rapidly progressing atrophy, with the associated phenomena of R.D.; without implication (in pure cases) of sensibility, micturition, and skin-nutrition; but usually with diminution of the reflexes,—of the tendon-reflexes more particularly.

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[1] Scheube, "Die japanische Kak-ke (*Beri-beri*)," Deutsch. Arch. f. Klin. Medicin, xxxi. and xxxii., 1882.

[2] Bälz, "Ueber Kak-ke (*Panneuritis endemica*)," Zeitschr. f. Klin. Medicin, iv. 1882. Mittheilgn. d. deutsch. Gesellsch. f. Natur- u. Völkerkunde Ostasiens, part 27, 1882.

The chief forms of disease of the anterior horns are the following. Acute atrophic spinal paralysis (poliomyelitis anterior acuta), often named spinal infantile paralysis, is a type of disease which calls for no remarks here, having been well characterised in its development, its course, its localisation, its sequelæ, and by a sufficient number of post-mortem examinations with uniform results.

The typical progressive muscular atrophy and its close ally amyotrophic lateral sclerosis are likewise characterised clinically by atrophic paralysis with R.D., anatomically by destruction of the anterior horns—the evidence being so clear as to leave no opening for any serious objection against their being set up as special forms of disease. It is clear, however, that the question may still be raised as to whether they consist in a true inflammation, or not rather in a slowly progressive primary degeneration; the future must decide upon this point. The form of atrophic paralysis which has hitherto been least investigated is the one known as Poliomyelitis anterior chronica (Paralysie spinale antérieure subaiguë, Duchenne) and which has been distinguished and defined on clinical grounds chiefly. The attempt made by Leyden[1] to reduce the spinal and peripheral atrophic paralyses to one group, and attribute to multiple neuritis the main part in the production of what has hitherto been understood as subacute or chronic poliomyelitis, does not appear to me very happy. Its result can only be to obscure a subject which was already undergoing the process of sifting and enlightening. Leyden falls into the very error which he censures in the supporters of poliomyelitis; a fact novel, or at least insufficiently studied, is thereby prematurely generalised and typified. It is certain that subacute or chronic multiple neuritis exists; and that many a case which has been described as poliomyelitic really belongs to it; but on the other hand, the results of post-mortem examinations[1] leave no doubt as to the existence of chronic poliomyelitis,—as a view strongly expressed by Eisenlohr (loc. cit.) and Remak.[2] Clinically, the two diseases may usually, in my opinion, be clearly distinguished, and if I may draw a conclusion from my own experience, the poliomyelitic type of disease is of far more frequent occurrence than is multiple neuritis.

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[1] E. Leyden, "Ueber Poliomyelitis und Neuritis," Zeitsch. für Klin. Medic. i. 887, 1880.

Chronic poliomyelitis consists in a more or less creeping and slow development of a paresis or paralysis with atrophy and R.D.; a paralysis which usually has a character of gradual ascent, without any disturbance of sensation beyond slight dysæsthesiæ, or paræsthesiæ, and moderate pains. These phenomena cannot be elicited by an objective examination, and are not accurately limited to the area of distribution of single nerves, but invade more diffusedly a whole extremity.

The distinction between chronic and acute anterior poliomyelitis rests mainly upon the slow development of the paralysis, its progress in time, and the more favourable prognosis; in the majority of cases the disease (unless it has an ascending subacute character) passes into a regressive stage, and a perfect recovery is obtained—a feature almost never present in the acute form, as Duchenne has already pointed out. This chronic form of atrophic paralysis does not seem to have hitherto been observed in children, among whom the acute disease is so prevalent. Recent literature of spinal diseases contains but negative statements with reference to this point, based on the general authority of Duchenne.[1] Thus Eulenburg,[2] Bristowe,[3] Ross,[4] and myself[5] have stated that the disease occurs only between the 30th and 50th year. Remak[6] says plainly that the disease is never met with in children. Ross himself, however, publishes a case,[7] with autopsy, of the disease in a girl of 15. Hughes Bennett[8] gives, under the name of poliomyelitis anterior chronica, an observation taken on a child 2 years of age, but which does not appear to me to belong to the category. (Disease beginning during measles; no marked atrophy of the paretic legs; knee-jerk present; electrical examination of legs not recorded.) The case of Ross would thus be that of the youngest individual affected; no certain instance has yet been recorded of the disease attacking a child.

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[1] Apart from the older results, see *Erb*, Krankh. d. Rückenmarks, 2nd ed., p. 722, 1883.

Aufrecht, "Ein Fall von subacut. Spinalparalyse," Deutsch. Arch. f. Klin. Medic. xxii. p. 83, 1878.

Bäumler, "Fall von Poliomyelit. ant. subac." Tagebl. v. 52 Versamml. d. Naturf. u. Aerzte in Baden, 269, 1879.

Edes. "A case of Anterior Spinal Paralysis with formation of vacuoles, etc.," Bost. Med. Surgic. Journ. 1879, July 24.

J. Ross, 'Diseases of the Nerv. System,' vol. ii. p. 139, 1881.

Fr. Neumann, "Poliomyelit. anter. subacuta, complicirt mit Leptomeningitis chron.," Deutsch. Arch. f. Klin. Med. xxviii. p. 589, 1881.

R. Schulz u. *Fr. Schultze*, "Z. Lehre von der acut. aufsteig. Paralyse," Arch. f. Psych. u. Nerv. xii. p. 457, 1881.

Landouzy et Déjerine, "Des Paralysies générales spinales à marche rapide et curable," Revue de Méd. 1882, Août.

C. Eisenhlohr, "Poliomyelitis anter. subacuta cervical. circumscripta," Mendel's Neurol. Centralbl. 1882, No. 18.

[2] *E. Remak*. Article: "Spinallähmung." in Eulenburg's Real-Encyclop. 1882.

I have lately made an observation on a child of six years old, which is calculated to fill this gap. Though recovery ensued, and thus the diagnosis has not received its confirmation by autopsy, yet it has been so carefully investigated with a special view to the differential diagnosis, that there cannot be any doubt as to the assumption of a chronic anterior myelitis being justified. In addition, the case is sufficiently interesting in itself, owing to the peculiar manifestation of its R.D., to call for an accurate description and commentary.

Clara Schellenberg, 6 years old, comes from a healthy family; mother slightly nervous. The child has had measles and hooping-cough, but otherwise has enjoyed uniform good health, and was always able to walk and romp like other children. Never had scarlatina nor diphtheria. Early in July 1822 the parents noticed that her right foot turned a little inwards, and was slightly dragged. About a fortnight afterwards the left foot showed the same symptoms, which very gradually became more marked, until the gait was greatly altered.

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[1] 'Electrisation localisée,' 3rd ed., p. 479.

[2] 'Nerven-Krankheiten,' 1878, vol. ii. p. 381.

[3] 'Theory and Practice of Medicine,' 3rd ed. p. 958.

[4] 'Diseases of the Nervous System,' vol. ii. p. 136.

[5] 'Rückenmarkskrankheiten.' [6] *Loc. cit.* p. 11.

[7] *Loc. cit.* p. 139. [8] 'Electrodiagnosis,' p. 121.

There never was any fever nor general disturbance; the disease was developed very slowly and gradually. Never any complaint of pains during the whole time. Micturition stated to have been a little delayed at first; but the function soon became quite natural again, and remained so. On the 22nd of August the child was brought to my policlinique (then in charge of Dr. Günther), and for the first time examined. The child, who was very intelligent, denied absolutely the existence of any pain whatever. No anomalies of the head, trunk and upper extremities. Both legs markedly paretical; the child cannot walk unless half carried, the toes hang down, the foot is put down awkwardly and unsecuredly; the gait is thereby very tottering. Below the knee there is distinct wasting. No perceptible alteration of sensibility; knee-jerk and plantar reflex absent. Functions of bladder and rectum perfectly normal. Electrical investigation shows partial R.D. in the district of both peroneal nerves. Direct galvano-muscular excitations yield sluggish contractions, but KCC is greater than A C C.

The child is galvanised twice a-week (current "stabile" to lumbar region, kathodic "labile" galvanisation of nerves and muscles of legs). The gradual invasion of the disease continues. Paralysis and atrophy increase. The child can no longer walk, but no pains are experienced.

On the 23rd of October, 1882, for the first time, I carefully examined the little patient. The child looks weakly, but is spirited and well developed. Her answers are to the point, and perfectly consistent. Head, trunk and arms normal, legs very weak, and locomotion greatly impaired, owing to complete paralysis of the peroneal districts on both sides; complete paralysis of the right tibial district; almost complete paralysis of the left tibial district (trace of flexion of toes left). Paresis of triceps extensor and flexors of thigh on both sides, slight on the left side, apparent on right. Functions of bladder and rectum normal.

Sensation unimpaired, as shown by repeated testing with reference to touch, pain, and temperature; the point and head of the pin are everywhere clearly distinguished. Tickling produces no impression on the soles, but neither does it on the upper part of the body. The farado-cutaneous sensation test, applied with my electrode, furnishes normal data, with no greater divergences than usually occur in patients of her age. No diminution worth mentioning in the lower as compared with the higher half of the body, nor difference between the two legs, especially in the districts of the paralysed nerves.

Plantar reflex and knee-jerk absent on both sides. The muscles are quite flaccid and soft. The joints passively moveable. The toes, when raised and abandoned to themselves, fall at once and quickly. Both legs are distinctly wasted: circumference of calf: right, 17; left, 18 cm.—of thigh (12 cm. above patella): right, 26·5; left, 27·5 cm. The muscles are not particularly tender to pressure, but their mechanical excitability is increased (especially in the peroneal district); the contractions obtained by their percussion follow a retarded course, and are tonic in character. In the vastus internus similar contractions obtained, but are rapid. The electrical exploration yielded peculiarly interesting results. I shall condense the results of accurate and repeated examinations of the peroneal district especially.

The faradic excitation of the nerves showed that the minimal contractions occurred at the following distance of the secondary from the primary coil.

N. frontal,	right,	206	mm.—left,	210	mm.
N. accessor,	„	227	„ „	220	„
N. ulnaris,	„	212	„ „	210	„
N. peroneus,	„	215	„ „	203	„
N. pro vast. int.	„	195	„ „	210	„

These numbers (considered along with the body-resistances estimated on the galvanometer) showed that there was no demonstrable quantitative alteration in the farado-excitability of nerves. The direct faradic exploration of the muscles gave :

Extensors of forearms :	right,	182 ;	left,	182
Flexors of forearm	„	190	„	190
Triceps extensor fem. (common motor point)	„	155	„	165
Vastus internus	„	170	„	195
Vastus externus	„	155	„	165
Tibialis anticus	„	163	„	160
Extens. digit et peron.	„	155	„	145
Muscles of calf	„	145	„	155

These numbers show a distinct, though not considerable diminution of the direct faradic excitability of the affected muscles.

The galvano-nervous exploration showed that the first K C O. occurred with the following number of cells and galvanometric deflections :

N. ulnar. ;	right,	12- (3°)—	left,	12- (3°)
N. peron.	„	12 (3°)	„	12 (3·5°)

Fourteen cells cause in the peroneal nerve very strong K C C, weaker A O C, very weak A C·C.

The galvanic exploration of the muscles showed in the vastus internus distinct K O C with 14 cells ; K O C > A C C, contraction not slow in this, nor in the other portions of the triceps extensor.

Marked R. D. in the anterior tibial, extensor digit. long., and peroneal muscles ; contractions very slow, tonic ; A C C>KCC ; no A O C. Latency of excitation very marked ; the contraction occurs late and lasts an unusual time.

In the calf-muscles excitability diminished, but not so tonic and protracted as in the peroneal.

The general result of this examination is, for the peroneal district, partial R. D., which in itself constitutes nothing strange. The remarkable feature about this case is the fact that the contractions had the characteristic protracted course and tonic duration, not only when obtained by direct galvanic excitation, but presented them also in an equally marked manner when obtained by the (galvanic or faradic) excitation of the nerves, and by the direct faradic excitation of the muscles.

This rare phenomenon was observed from the very first exact observation made, but was submitted repeatedly to various tests, the results of which were shortly as follows:—The galvanic excitation of both peronei with 12–14 cells (the duration of the closure being only momentary) is followed by a lazy, tonic contraction, which passes off gradually,—not so protracted, however, as that due to direct muscular excitation. The contraction obtained by AO is likewise slow and persistent. On increasing the strength of the current, the line of ascent of the contraction increases in slope and height, but that of descent is always gradual (as is well shown by the contrast with the sudden fall of the foot from the state of passive dorsal flexion). Faradic excitation of both peroneal nerves (secondary current and rapid vibrations of the hammer, either A or K of break-current) produces the same abnormal course of contraction even when the circuit is closed for a very short time. Powerful currents cause a powerful and rapid rise of the muscle, but on opening the circuit the relaxation is still characterised by its sluggish course. The primary (extra) current gives rise to the same phenomena. In order to ascertain whether instantaneous excitations had the same effect, single induction shocks (of secondary or primary) were tried, and found to be followed by contractions having the same gradual rise and fall.

The faradic stimulus directly applied to the muscles themselves gives—if somewhat stronger than when applied to the nerves—the characteristically developed contractions. The alterations are much more accentuated than in the contractions elicited by indirect excitation, and are observable whether rapidly interrupted currents or single shocks are used.

With reference to the order of contractions, repeated observations show that nerves react only to KC and AO (KCC > AOC; contraction slow), and that muscles react only to KC and AC (ACC > KCC; contraction much protracted and slow).

I shall now give a brief summary of the progress of the case, which went on very satisfactorily. On November 3rd, 1882, the first traces of returning motility in the peroneal district were observed, all the other circumstances, including the electrical phenomena, remaining the same.

November 27th.—Amelioration marked. Motion of left foot extensive and forcible; distinct movements on the right side in the peroneal and posterior tibial districts. The child moves about easily. Triceps extensor and flexors of thigh much stronger, on left side especially. Alteration of contractions still very marked on right side, much less so on the left. Circumference of calf: right, 18 cm.; left, 19 cm. Sensation always perfect; no pain.

December 9th.—The left leg is almost normal in its movements; the knee-jerk has reappeared in it, but not on the right side. Right foot moves better. Electrical contractions approximate the normal type on the left side: 12 cells, slow A C C: 16 cells, rapid K C C. On the right side the anomalous responses still persist.

December 31st.—Continuous, progressive improvement. On the left side the condition is almost normal. Muscles fuller, knee-jerk lively; farado- and galvano-nervous and muscular excitations do not call forth delayed contractions. A O C recedes into the background. On the right: motility much improved, especially in the peroneal district. Muscles still flaccid; no knee-jerk. Electro-muscular excitability diminished, but still protracted responses to both direct and indirect excitation.

January 19th, 1883.—Further progress. Left leg quite normal. Right still weak, drags readily; still the child goes about in a lively manner; knee-jerk absent. Electrical phenomena returning to the normal; contractions much less protracted. A C C still prevails slightly in muscles. Faradic excitability of both peroneal nerves equal (203 mm.). Farado-cutaneous exploration gives the same results as formerly. Muscles firmer and fuller. Circumference of calf: right, 18·5 cm.; left, 19·5 cm. Thigh (12 cm. above patella): right, 28 cm.; left, 29 cm. General condition excellent; feet less cold.

Commentary.—The diagnosis of this case—unique, *quoad* the age of the patient—cannot be doubtful in my judgment. We have to deal with an atrophic paralysis of gradual and progressive development, and with partial R.D. in the lower extremities, without the trace of any anæsthesia, paræsthesia, or pain; a paralysis which does not consist in the successive implication of the nerve-trunks involved, but which invades the whole lower extremity in more diffuse and with diminishing intensity as it ascends upwards. The sphincters are free; the general state is not disturbed. It presents, therefore, the typical image of a spinal atrophic paralysis; a multiple neuritis is excluded by the absence of every sensory disturbance and the diffuse distribution of the paralysis. The most obvious supposition is that of a lesion (inflammatory ?) of the anterior horns of the lumbar enlargement, most severe in the lower half of it (nuclei of origin of the sciatic nerve), and diminishing in intensity as it ascended into the upper half (nuclei of the lumbar plexus), the right side being more deeply affected than the left. Such is the disease which we call chronic anterior poliomyelitis. The case before us belongs more particularly to the class of milder cases forming the "middle form" of poliomyelitis previously described by myself;[1] but I should no longer consider it necessary to give a special description of this form, which obviously is but a variety of a single morbid type.

The favourable course of the disease, culminating in complete recovery, is an important characteristic element of it; a course frequently observed among adults, in opposition to the acute type of poliomyelitis, or infantile paralysis, from which it may be diagnosed by the partial R.D. and this favourable course, as well as by the kind and mode of its development.

The result of the galvanic treatment, the only one employed, deserves some remarks. Whether we must ascribe to it the fact that the left leg, which was the last attacked, recovered first, is of course not easy to determine; and this observation cannot be considered as a proof of the value of galvanisation. Still such a possibility deserves to be noted.

Of greater interest still is the peculiar behaviour of the partial R.D. in the nerves and muscles involved. The main point is that every kind of direct and indirect excitation of the muscles, even instantaneous stimuli, invariably produced sluggish contractions. This is usually not the case with partial R.D., such contractions occurring only upon direct galvanic excitation of the muscle; its faradic excitation, like its indirect excitation by either current through the nerve, yields only the normal rapid, short-lived, contraction. But all this was altered in our case.

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[1] Erb, "Ueber eine noch nicht beschriebene 'Mittelform' der Chron. Atr. Spinallähmung." Centrlbl. f. Nervenheilkunde, I. 3. 1878.

These phenomena, however, are by no means absolutely new, they have been previously observed. I have myself in a paper on Peripheral Paralysis[1] described a case of ulnar paralysis in which faradisation of the muscles, and later of the nerves, called forth a distinctly retarded contraction. E. Remak[2] observed the same phenomena in a case of subacute atrophic paralysis, and has given to this sub-species of partial R.D. the name of "Faradic R.D."

Two of my former assistants[3] have related cases of neuritis which occurred under my own observation in which this peculiarity occurred. In one of the cases the contractions were lazy when obtained by galvanic excitation of the nerves.

Kast[4] has also described quite lately a case in which a certain diagnosis (peripheral or poliomyelitic lesion) could not be arrived at, but where the same anomaly was found. The contractions obtained by faradisation of nerve or muscle were protracted. The galvanic excitation of the nerve, however, appears to have been followed by rapid and short-lived contractions.

Finally, I have observed the phenomena in several cases of paralysis of various origin ("rheumatic," in facial; traumatic, in median; pressure, in musculo-spiral); it was more or less accentuated, occasionally mere traces of it were present. It would thus appear not to be so very rare, and I am inclined to think that careful investigation will prove it to be less rare still, especially as a temporary or transitional condition in the course of the disease.

With reference to the question as to the meaning of this peculiarity in the partial R.D., I can but subscribe to the remarks made by Kast (loc. cit.). We have not to do with a fundamental departure from the typical course of R.D., but, apparently, with a mere deviation in those histological changes of the muscles which are doubtlessly at the root of the altered reactions. In the partial R.D. these alterations are usually insufficient to destroy the normal rapid wave of the neuro-muscular contraction; in certain cases they go so far as to change this contraction into a long-drawn one. The direct excitation of muscle by faradism is, we must remember also, of the same order as the indirect muscular one. This theory has nothing to militate against it, though of course it is not an easy task to give a histological proof of its truth. As Kast happily observes, our variety of R.D. may form a connecting link between the complete and partial forms.[1]

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[1] Deutsch. Archiv f. Klin. Med. 1868, iv. pp. 554, 556. See also my 'Elektrotherapie,' p. 210.

[2] Arch. f. Psych. u. Nervenkrankheiten, 1879, ix. p. 551.

[3] Kast, "Zur Lehre von der Neuritis." Arch. f. Psych. 1881, xii. p. 266. Vierordt, "Ueber atr. Lähmungen der ob. Extremitäten." Deutsch. Archiv, 1882, vol. xxxi. pp. 495, 501 (cases 4 and 10).

[4] Neurol. Centralblatt, 1882, No. 17.

I do not now think it so justifiable to call it "faradic R.D.," as Remak has proposed to do, since we find that indirect galvanic excitation may also be followed by long-drawn contractions. The main point, I repeat, is the occurrence of the abnormally slow muscular contractions to both kinds of electrical nerve-excitation, and to faradic muscle-excitation; hence I would propose to call this variety "the partial R.D. with indirect slow contraction" ("mit indirecter" oder "mit obligater," Zuckungsträgheit).

With reference to the clinical and prognostic meaning of this form, Kast is right in concluding from its occurrence to the presence of a change of moderate intensity and hopeful issue in the neuro-muscular apparatus. The cases in which it occurs may be expected then to run a favourable course.

These observations teach us that we have not yet exhausted the list of the numerous manifestations of R.D., and that the histological changes which are at the root of it may present numerous modifications of which we are as yet unable to give a connected account or explanation.

[A. DE W.]

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[1] Cf. Erb, 'Elektrotherapie,' pp. 178 ff.; 197 ff.

ON EPILEPSY, IN ITS RELATION TO EAR-DISEASE.

BY J. A. ORMEROD, M.D.

It needs no very extended observation to show that these two phenomena frequently co-exist. In such a case there are several modes of relation possible: either they are simply coincident, or both are the effects of some common cause, or the one is the cause of the other, the epilepsy of the ear-symptoms, or the ear-disease of the epilepsy. It will be my object to discuss these various relations, together with some subjects of interest that suggest themselves in connection with them.

The term ear-disease I shall use with considerable latitude, wishing to consider on the one hand the production of aural symptoms only in the course of a simple epilepsy, and on the other the possible production of epilepsy by a definite aural disease, viz. purulent otitis.

Instead of attempting a regular definition of the term epilepsy, it will be sufficient perhaps to make a provisional grouping of chronic convulsive disorders, and indicate that kind to which I refer. We may distinguish as follows: First, the "hysteroid" fit. The patient is usually a female or a boy. The coma is not so deep but that the patient can be, to a certain extent, roused from it; nor so sudden in its onset but that she avoids hurting herself as she falls. The convulsions are not limited to mere spasms, but co-ordinated movements occur, such as biting, striking at the bystanders, resistance to restraint. Secondly, the epileptiform fit. In this, coma is either absent altogether or comes on late in the attack; the convulsions are apt to spread slowly from some given point, and may be limited to one side; not unfrequently there is double optic neuritis, and post mortem, some localised structural disease is found. Both these types of fit I have endeavoured in the present paper to exclude. The third kind, which I shall provisionally call epilepsy proper, and to which I limit myself, may be roughly characterised as follows. There may indeed be "warnings," but the coma itself comes on suddenly and early in the fit, and is profound. The patient falls anywhere, often injuring himself. The tongue is often bitten. The convulsions, as a rule, spread rapidly, and are usually bilateral. They may, however, be very limited or very slight. Minor attacks may occur, consisting of the "warning" only, or of transitory coma without perceptible convulsion (petit mal). If paralysis occur, it is incomplete, transient, and immediately consequent upon a fit. There is no optic neuritis, and no definite anatomical lesion can be predicted.

It cannot be denied that in some, perhaps in many cases, the co-existence of epilepsy and ear-disease is merely accidental. The greater the absolute frequency of the two diseases, the higher does the probability of such a relation become, and it is difficult to exclude unless some causal connection can be shown to exist.

Or again, the two diseases may be connected, not by mere accident, nor in the sense that one causes the other, but that they both have a common parentage. The effects of scarlet fever are an example of this. Scarlet fever, by extension of the throat-affection to the tympanum, produces, as is well known, many and severe cases of middle-ear disease. It may also, as Dr. Gowers[1] has suggested, produce epilepsy; an effect due, he thinks, to some peculiar action of the poison on the nervous system. It is tempting where both ear-disease and epilepsy are left after the fever, to regard the ear-disease as the link between the epilepsy and the fever; but this is not always so, for in only one out of Gowers's nineteen cases of epilepsy after scarlet fever did ear-disease appear. It may therefore be fairly said that in some cases epilepsy and ear-disease may result from scarlet fever directly and independently of each other. We may ask whether other acute specific diseases, in which the throat is severely affected, as for

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[1] 'On Epilepsy and Chronic Convulsive Diseases,' p. 28.

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instance diphtheria, may not sometimes produce the same effect? Acquired syphilis appears to produce deafness in exceptional cases, and sometimes epileptic fits, either of the Jacksonian type, or else, according to Fournier, indistinguishable from those of ordinary epilepsy. In congenital syphilis, on the other hand, deafness is well known, and it seems probable that some cases of epilepsy at an early age may be ascribed to this cause. The two following cases are offered in illustration of this:

A. B., æt. 12, was brought to me at Queen Square Hospital in March 1882. He had incisors of the type described by Hutchinson; the right cornea was dim, the left sclerotic injected in the neighbourhood of the cornea. He had been subject to twitchings of the limbs for eighteen months, and, during the last six weeks, to fits, with loss of consciousness. A sister, about 15 years old, had also characteristic incisors, and had had fits from birth. The mother had had six miscarriages. The mother's brother had also recently developed fits. This last fact makes it probable that the syphilitic taint was not the only agency at work: but in the next case there was no such history of epilepsy in the previous generation.

A. F., æt. 9, had had fits for four years; at first they had been "like faints," accompanied by sleepiness and vomiting, and preceded by loss of appetite: during her attendance they changed to sudden falls, followed by general convulsions. She had the characteristic incisors; there was disseminated choroiditis in both eyes, and she had one or two attacks of keratitis while attending. There was in this case no deafness. The mother had had four miscarriages. A sister, 4 years old, suffers from vomiting, is rather wanting in intelligence, and is supposed to have had a fit.

We turn now to the question, How far can epilepsy be the cause of aural affections? It is not of course asserted that structural diseases of the ear, such as perforations of the membrane, disease of bone, inflammation or chronic thickening of the tympanum, can be so caused; but that certain subjective symptoms commonly met with in aural practice, which must ultimately, like all subjective symptoms, be referred to the nervous system, may occur in the course of epilepsy uncomplicated, so far as can be ascertained, by disease of the ear. Such symptoms are pain in the ear, noises in the ear, deafness; and on the other hand, vertigo, rotatory movements or other disturbances of equilibrium, vomiting.

Some epileptics complain of pain in the ear, apart from any aural disease, but I imagine that this can usually be recognised as part of the general headache to which they are so prone, analogous perhaps to the pain of megrim when localised in the eye. Let us, however, note the fact that the pain of actual tympanic inflammation is relieved, like the epileptic headache, by bromide of potassium.

Noises in the ear are due to various causes. Apart from those that are really not subjective, but correspond to an actual physical phenomenon, e.g. the bruits of anæmia and of aneurism, noises may be caused by wax in the meatus, catarrh or chronic thickening of the tympanum, &c.

Irritation of the auditory nerve appears to be the chief modus operandi of these causes; in fact Erb [1] maintains that tinnitus in some cases is due solely to hyperæsthesia of the auditory nerve, and that such cases may be distinguished from others by the curative effect of the galvanic current properly applied. A subjective sound in connection with epilepsy usually occurs as an aura. Since such an auditory aura is held to be rare,[2] I give the following instances:—A woman, æt. 27, epileptic for five years, said that, as the fit came on, she had sometimes a ringing noise in the ear, not always in the same ear apparently. She then fell unconscious; she had bruised herself and bitten her tongue.—A man, æt. 32, had as warning "an indescribable sensation" starting from his big toes, and usually noises in his ears.—Another man had a hissing noise in his ear before the fits.—A fourth had occasionally a "ringing;" a fifth, "a drumming feeling" in the ear, independently of the fits. In all these cases the ears were normal. Where the ears are diseased, noises of course may be caused by the ear-disease only; yet sometimes even then they seem to occur in close connection with the fits. Thus a woman with slight and variable impairment of hearing, and subject to a noise in the left ear, which was aggravated by stooping, said that her first two fits "were preceded by noises like rushing water in that ear." Again, a man whose ears were partially filled with wax, said that as an aura he used to have a humming noise in his ears, accompanied by strange sights before his eyes. Possibly such facts may point to a connection between the peripheral and the central disease. From cases of auditory aura we may at least infer that, if as is generally held, the phenomena of the epileptic fit are due to "discharges" in the cells of the cerebral cortex; the sensation of sound usually caused by some disturbance, natural or morbid, of the auditory nerve or its peripheral expansion can be caused also by processes originating in the cortex. Experimental evidence of an auditory centre in the cortex has been offered by Ferrier and others. Pathological illustration of the connection between the action of the auditory nerve and that of the cerebral centres may be sought in the cases where the mere tinnitus of aural disease becomes transformed into aural illusions. Thus a woman, æt. 61, had been epileptic for thirteen years, and deaf for a longer period. Lately, however, her deafness had got much worse, she had noises in the head, and attacks of vague vertigo. The condition of the ears (the membranes were depressed and thickened) seemed enough to account for the aural symptoms, though her inability to hear the tuning-fork through the skull made it probable that the labyrinth or the nerve had also suffered. The tinnitus increased steadily, till she began to hear not only noises, but words, names of streets, songs, &c. She was said to be irritable, but seemed perfectly sane, though subsequently illusions of smell and vision developed.

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[1] 'Elektrotherapie,' p. 231.

[2] Hughlings-Jackson, 'British Medical Journal,' 1877, vol. i. pp. 386, 703; and 'Lancet,' March 11, 1876.

Roosa[1] quotes the opinions of Schwartze, that "subjective aural sensations, which are caused by demonstrable affections of the ear, may in predisposed persons, especially when there is any hereditary tendency to mental disease, become the direct cause of aural hallucinations, that may accelerate the outbreak of a disease of the brain."

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[1] 'Treatise on Diseases of the Ear,' p. 265.

The opposite affection, deafness, may also occur in the course of epilepsy. With regard to infantile convulsions, Itard affirms this in very positive terms. Speaking of what he calls "paralysis of the acoustic nerve following convulsions,"[1] he says, "this cause of deafness is very rare in adult life, and very frequent in early life. When hearing is lost in the first three or four years of life, it is nearly always a consequence of convulsions. A great number of deaf mutes owe their infirmity to this cause, which, destroying the hearing at a very early age, finally entails the loss of speech. It is worth remarking that it is commonly the less violent and less prolonged convulsions which produce this effect. Numbers of infants who had become deaf about the time of dentition, and for whom I had been consulted, had for the most part ceased to hear suddenly, directly after a convulsive attack." Itard himself does not illustrate these remarks, but six cases of the kind are given by Knapp.[2] Arguing from the sudden outset and the complete character of the deafness, Knapp suggests that a serous or hæmorrhagic effusion into the labyrinth causes, on the one hand the convulsions by reflex irritation of the medulla, on the other, the deafness by destruction of the labyrinth. I cannot myself call to mind more than one case of this kind out of a large number of epileptics who have had convulsions in early life; and Knapp himself remarks that "most writers on otology say little or nothing on the subject." But there appears to be a different kind of epileptic deafness, viz. a deafness which may be slight, unilateral, occurring after the fit, or intensified then, and sometimes connected with an auditory aura. A young woman, æt. 19, had had fits in infancy, chorea when 8 years old, and fits again during the last two years. There was no warning; she had occasionally bruised her face and bitten her tongue. After the fits she said a pain went through to the left ear, and she felt rather deaf. The tympanic membranes were normal. A watch (the distance for hearing which was 4 feet or more) she could hear through 4 feet with the right ear, through 40 inches with the left. The tuning-fork applied to the middle line of the cranium was heard best in the right ear (she was quite positive of this). On one occasion she had had a curious attack; without losing consciousness she turned round and round from left to right, and fell against a table.[1] Another woman, æt. 35, had had fits since her first pregnancy, fourteen years ago, and fainting fits before that. In the fits she bit her tongue and lips, afterwards she felt "lost and silly." Since the fits, she has been quite deaf with the left ear, having been slightly deaf with it for some years previously. Inasmuch as she stated that she had always giddiness and a hissing noise in the head, it seemed probable that she had aural disease, but I could find no evidence of it; the tympanic membranes were normal, and the tuning-fork, whether in the air or upon the cranium, was only heard with the right ear. Moreover, when the fits were stopped by treatment, the deafness certainly improved; she could then hear the watch ½ an inch from the left ear. Two other cases, of which, however, I have no satisfactory aural examination, seem somewhat similar. The one, a single woman, æt. 30, said that after each fit she was partially deaf chiefly with the left ear; she felt a "sort of numbness" of the ear. The other, a man, æt. 21, gave the following account—"it" (i.e. the sensation constituting the aura) seemed to rise up from the toes and leg of the right side into the right ear; he became deaf with that ear at the time, and remained so for two or three days after the fit.

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[1] 'Traité des maladies de l'oreille.' Tom. ii. p. 315.

[2] 'Archives of Ophthalmology and Otology,' vol. ii., No. 1, p. 260.

I put forward these cases with much reserve, because it is difficult for any but a skilled aurist to exclude with certainty disease of the middle ear.[2] But analogy seems to show that there may be such a post-epileptic temporary deafness dependent upon a morbid condition of the cortex cerebri. Epileptic patients will sometimes complain of attacks of blindness, while the ophthalmoscope shows the optic nerve to be healthy. Then there are cases of post-epileptic paralysis of the limbs, incomplete and transient (in cases of pure epilepsy), affecting the limbs which have been most convulsed, and dependent (as has been shown in cases of gross lesion) upon disease of a corresponding cortical area. Moreover, there are post-mortem records of cortical lesions where there had been deafness during life.[1] But unfortunately we cannot do much, clinically, towards localising the various forms of nervous deafness. Not only are the anatomy and physiology of the various apparatus of hearing less well known than those of vision, but our means of clinical investigation are much more limited in regard to the ear. Perhaps the symptom insisted upon by Knapp,[2] viz. deafness for certain groups of tones, may suffice for diagnosis of disease of the cochlea, or perhaps electrical investigation may afford some clue to the condition of the auditory nerve;[3] but these methods, to say the least, necessitate the intelligent co-operation of the patient, and cannot make up for the lack of an instrument like the ophthalmoscope for the direct inspection of the nerve and its expansion. Even the simple diagnosis by the tuning-fork of nervous deafness (i.e. deafness due to lesion of the internal ear and parts beyond) from middle-ear deafness, rests upon information derived from the patient.

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[1] This vertiginous attack and the slight internal ear-deafness suggest the possibility of Menière's disease; there was, however, no tinnitus, and the deafness which the patient connected with the epilepsy, was not progressive.

[2] Since writing this, I find a case given by Knapp, 'Archives of Otology,' vol. xi., No. 3, p. 232. A man, æt. 37, had become permanently deaf after epileptic fits; the middle ear and tympanic membranes were normal.

In close connection with the organs of hearing, both by anatomical and pathological relation, stand the organs of equilibrium. If not justified in speaking of a special sense of equilibrium, we are at least conscious of perturbation of that sense, the feeling expressed as vertigo. Vertigo is defined by Brunner[4] as "an impairment of the sense of equilibrium," and described by Ramskill[5] as "the sensation of moving, or the appearance of moving objects, without real movement." It is worth while considering some of the conditions under which this sensation arises. The peripheral organs from which we derive a knowledge of the position of our bodies in space, appear to be of two kinds. First, organs which seem to have a purely sensory function, and which enable us to appreciate the effects even of passive movements apart from the other senses. These are mainly the semicircular canals. Perhaps certain mobile abdominal viscera may contribute to the same end. Secondly, a combination of the muscular sense with the special senses of sight and touch. Retinal impressions per se give us little information as to the position of objects. To judge of their distance from us, and of their position above, below, or to either side of us, we must appeal to the muscles which accommodate the eye and move the eyeballs. A similar combination of touch with muscular effort aids us in the absence of the sense of sight. Thus a patient with locomotor ataxy, whose muscular sense is impaired, finds it difficult to stand with his eyes shut; but the difficulty is very much increased if the soles of his feet have become anæsthetic. Whether the sense of hearing can enter into any such function appears extremely doubtful; the other senses certainly do not.

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[1] E.g. cases quoted in 'BRAIN,' October, 1882.

[2] *Op. cit.* p. 255.

[3] Erb, *op. cit.* p. 226.

[4] 'Archives of Ophthalmology and Otology,' vol. ii. No. 1, p. 293.

[5] 'Reynolds's System of Medicine.' Article on Vertigo.

When the report of the special senses and of the muscular sense are inharmonious, vertigo is the result. This is well seen in the case of ocular vertigo.[1] A man with paralysis, say of the right external rectus, is giddy when, the sound eye being closed, he looks to the right. The motor effort required to fix objects in that direction is greater than, under normal circumstances, is necessary: hence, so far as this is concerned, he places objects to the right of what they should be; as will be seen, if he tries to put his finger quickly on any small object. But inasmuch as his other senses contradict this neuro-muscular report, vertigo is the result. In this case the motor effort is in excess of the effect produced. The reverse relation may hold. In the case of involuntary muscular spasm, e.g. the rotatory movement which precedes many epileptic fits, the motor effort (or at least the volitional part of it) is insignificant as compared with the result; hence the vertigo. In view of the important part played by the muscles with reference to equilibrium, it has been said that the physical basis of vertigo is an incipient motor process.[2] The sensation of vertigo is the representation of such a process in consciousness. In the vertigo produced by disorderly action of the semicircular canals, as in the experiment of whirling a person round with his eyes shut, it is not so clear that the muscular sense plays any part, though it is true that in severe paroxysms of aural vertigo compensatory muscular movements do take place.

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[1] Hughlings-Jackson, 'British Medical Journal,' 1877, vol. i. p. 605.

[2] The phrase applies not so much to the actual muscular contraction, as to the nervous current which causes it.

With respect to the co-ordinating mechanisms by which equilibrium is actually maintained, it is to be remarked that in the lower animals at least it can continue acting after the removal of the cerebral hemispheres, and therefore in a reflex fashion, and independently of consciousness. This is shown by Goltz's balancing experiment. (Are there not indications of the same fact in the hysteroid fit, where, notwithstanding the apparent, and I think I may say real, loss of consciousness, the patient so adjusts herself as not to hurt herself in falling?) Nevertheless, we should imagine that the superadded state of consciousness which we call vertigo, must correspond to changes in some area of the cerebral cortex, if physical changes in that part accompany consciousness upon the psychical side. And considering the numerous and important character of the organs concerned in the maintenance of equilibrium, it is likely that this area would be large. Assuming, therefore, that epileptic vertigo consists in morbid action within such area, as epileptic spasm in the motor area, sensory auræ in the sensory area, &c., we should expect vertigo to be a frequent phenomenon in epilepsy. That it is so is a well-known fact, which I need scarcely stop to illustrate. But it is not always easy to arrive at the fact in any given case. The patient usually complains of giddiness. But that expression, as has been remarked by Dr. Hughlings-Jackson, ought not to be taken as equivalent to vertigo. It may mean a number of vague sensations, such as faintness, and the like; and many patients cannot analyse their feelings further, unless we put the leading question, whether things seem to move, when, of course, the affirmative answer is less valuable than if it were volunteered. Nevertheless, there are many who will voluntarily describe a sensation of vertigo, and many more, I suspect, who have such feelings, but cannot express them.

However, the point which in the present place I wish to insist upon is, that vertigo, though corresponding to action of the cerebrum, may be occasioned by disease in very different parts, either in the cerebrum itself, as in the case of epileptic vertigo, or in a peripheral organ, the commonest being the semicircular canals. This latter kind is aural vertigo. The semicircular canals may be actually diseased, as we suppose them to be in Menière's disease, or affected secondarily and as to their function only, as may happen in middle-ear disease. The explanation usually given of this latter fact is that the middle-ear disease causes increased pressure in the labyrinth,[1] and possibly also increased excitability of the auditory nerve. Such vertigo may be distressing and alarming to the patient, but a knowledge of its peripheral origin may enable us to assure him that it is unimportant, at least as regards life and mental functions. A well-known aural text-book says:[2] "When vertigo occurs in aural disease, it is a consequence of increased pressure upon the labyrinth through the fenestra ovalis. It is by no means a serious symptom when the cause is to be found in the middle ear, for it is usually relieved by a mechanical treatment through the Eustachian catheter."

Clinically associated with vertigo is the symptom of vomiting. Every one who has been sea-sick will admit its connection with perturbation of equilibrium. Vomiting often concludes a fit of convulsions (especially in children); or concludes, on the other hand, an attack of aural vertigo. In the latter case the explanation usually offered is that the irritation overflows, so to speak, from the auditory into the adjacent pneumogastric nucleus. Indeed, irritation of the pneumogastric appears to be the proximate cause of vomiting generally (Ferrier). There is no doubt that vertigo may produce vomiting, but can gastric derangement produce vertigo? Such a "gastric vertigo" has been forcibly described by Trousseau, but recent writers appear either to doubt its existence, or at most to admit that gastric derangement may occasion vertigo in the presence of aural disease. Theoretically, if we suppose that irritation can spread from the auditory to the pneumogastric centres, then it does not appear why it should not also spread the other way. It may be remarked that Trousseau, though he does not mention the state of the ears in his patients with gastric vertigo, was well aware of the existence of aural vertigo, which he describes in the same chapter.[1]

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[1] Knapp, *op. cit.* p. 214; Brenner, *op. cit.*

[2] Roosa, p. 264.

We may ask whether diarrhœa ever takes the place of vomiting in aural vertigo? It appears to do so in some nervous affections. I have known a successful struggle against sea-sickness to be followed by diarrhœa the next day. A man, recently under my observation for locomotor ataxy,[2] who suffered from the well-known "crises gastriques," had also unaccountable attacks of diarrhœa; the same thing sometimes occurs in epileptics, while, reversely, bromide of potassium occasionally constipates.

I append brief notes of a case in which the symptoms of vertigo, vomiting and diarrhœa seemed distinctly to take the place of epileptic fits, the ears being quite healthy:—

William H., æt. 15, became an out-patient at Queen Square Hospital, in June 1880. He had had fits for twelve months, undoubtedly epileptic. There was no warning, he had bitten his tongue, in the first fit he had fallen and cut his face. He was also subject to "startings" of the hands and feet, without loss of consciousness; these "starts" had preceded the outset of the fits by some three months. Under bromide of potassium and arsenic the fits were stopped.

Feb. 1881.—Nausea and abdominal pains, on account of which the arsenic was dropped.

April.—"Swimmings in the head."

May.—Giddiness, a feeling as if he must fall, obliging him to sit down; nausea also.

June.—Has been vomiting. Alternate diarrhœa and constipation. . . . Later in June, definite vertigo, "the room seems to turn from right to left." The hearing, according to his statements and as measured by the watch, was normal.

Hydrobromic acid relieved all his symptoms (except slight giddiness) during the rest of the year.

January 1882.—Severe attacks of vertigo, retching and vomiting. . . . Hearing (watch and tuning-fork) normal. Tympanic membranes normal. (Same results later on.)

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[1] Syd. Society Translation, vol. iii., Lect. 67.

[2] *Cf.* Buzzard, 'Clinical Lectures on Diseases of Nervous System,' p. 199.

During the first half of the year the symptoms abated again, but he had some "faints."

July.—Has had a bad attack of giddiness, vomiting and diarrhœa: the diarrhœa persists, and appears to be made worse by the hydrobromic acid. It was changed for bromide of potassium.

August.—One fit. Diarrhœa comes and goes. Occasional giddy attacks; no vomiting.

October.—After the attacks of giddiness, now becomes extremely sleepy: once slept soundly for three hours. Their connection with the epilepsy is thus made all the more manifest.

So far we have considered certain symptoms common in aural disease (meaning by that term chiefly middle-ear disease) and have endeavoured to show that they may occur to a greater or less extent in simple epilepsy. But they are the symptoms which characterise a complaint which may be fairly said to stand midway between these two, i.e. the affection known as Menière's disease. It will be asked, since these symptoms occur in middle-ear disease, what are the clinical characteristics of the so-called Menière's disease? A consideration of Menière's original cases (Note A), shows, I think, that in most of them, at least, the paroxysms of vertigo and vomiting are more sudden, more violent, more definitely paroxysmal than in chronic middle-ear disease, and that the "vital symptoms," as they have been called by Dr. Hughlings-Jackson,[1] viz. perspiration, pallor, faintness carried even to coma, are more marked. Secondly, the deafness is of the kind known as internal ear-deafness, i.e. inspection of the ears yields negative results, and sounds conducted through the cranial bones, as well as aerial vibrations, are heard imperfectly or not at all with the affected ear.

But since the vertigo of middle-ear disease may be paroxysmal and severe,[2] and since there may be cases of Menière's disease which in the early stages exhibit little if any impairment of hearing, further distinctions are necessary, and these seem to lie (as Dr. Pritchard suggests to me) in the course of the symptoms. In Menière's disease, pari passu with the repetition of the paroxysms, the tinnitus and the deafness increase, till at length the patient is left free from the vertigo and other symptoms, but hopelessly, perhaps absolutely deaf.

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[1] "On Nervous Symptoms with Ear-disease." 'British Medical Journal,' March 1877.

[2] Knapp, *op. cit.* pp. 218, 241; Brunner, *op. cit.* p. 319; Trousseau, *op. cit.* p. 552.

The differentiation from cerebral disease was made by Menière himself, and based partly on the occurrence of tinnitus and deafness (Note A), partly on the post-mortem evidence of his last case. With respect to this case it has been remarked that, though valuable for purposes of localisation, it does not give us much information as to the pathological process in the typical disease. In the typical disease there is a series of paroxysms, perhaps persistent impairment of equilibrium, but ultimate destruction neither of life nor of any function except hearing. The case in question consisted of a single paroxysm with rapidly fatal result.

To recur to the connection with cerebral disease. Seeing that the morbid anatomy of many grave nervous diseases is yet to seek, we cannot from the negative results of a post-mortem infer that the nerve-trunks or nerve-centres have not been essentially affected during life. Indeed, the "vital symptoms," faintness, sometimes sudden coma, &c. (in one of Menière's cases there were even convulsions) — make it certain that the higher centres are engaged, if only secondarily. The paroxysmal recurrence of the symptoms points in the same direction. It is possible that a close inquiry into the family and personal history of the patients affected with this disease may disclose some inherent weakness of their nervous system. Dr. Gowers[1] suggests "that if we locate the centre for equilibrium in the cerebellum, we may perhaps regard Menière's disease as a cerebellar epilepsy reflex in character, determined by an irritation of the semicircular canals, either alone or in conjunction with impressions from other nerves with which the centre is in connection."

The question whether ear-disease can contribute to the production of epilepsy is evidently one of some practical importance. Although most writers on epilepsy do not allude to it, there seems to be a certain à priori probability of such a relation. "Excentric" causes of epilepsy are generally admitted to exist; and it is not unnatural to place ear-disease among them, considering on the one hand the nervous phenomena (such as we have already discussed) which may be produced by the irritation, even of non-suppurative ear-disease, and on the other the fact that such gross diseases as cerebral abscess and meningitis may result from a neglected otorrhœa. Limiting the discussion to the subject of suppurative ear-disease, I shall quote what Dr. Hughlings-Jackson says on this subject:[1] "Epilepsy, or epileptiform seizures, occasionally occur with ear-disease. Admitting a relation, the accepted explanation would be, that the ear-disease provokes the seizures by reflected irritation. The author suggested another hypothesis, the possibility that the aural disease led to disease in Hitzig's and Ferrier's region; for in some cases, the aural disease was associated with epileptiform convulsions, starting in the hand, face, or foot of the opposite side. In one such case, the author had found a large tubercular or scrofulous tumour of the hemisphere on the side of the ear-disease, and on the side opposite the convulsions. He thought it possible that the disease is sometimes local softening from venous thrombosis, the convulsions depending on instability of grey matter around the part softened. He had no direct evidence of this, but he thought he had some indirect evidence of it. He related[2] a case in which a man who had phosphorous necrosis of the jaw had, during an acute illness, frequent epileptiform convulsions affecting the left side of his face, both sides of the thorax, and slightly the left arm. In the intervals of the attacks there was paralysis of the face almost as complete as in ordinary Bell's paralysis. At the necropsy there was, although not ear-disease, yet an equivalent state of things, there being pus in the right lateral sinus, and from this a vein with creamy contents was traced to the part most diseased of the right side of the brain; about the anterior end of the Sylvian fissure there was purulent softening and red softening. As the lesions were more wide-spread, and as the case was acute, the evidence was only very indirect towards the interpretation of chronic cases, but was, the author thought, worth consideration in that regard."

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[1] "On Auditory Nerve Vertigo." 'British Medical Journal,' March, 1877.

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[1] "On Nervous Symptoms with Ear-disease." 'British Medical Journal,' March 24, 1877.

[2] This case is recorded: 'Medical Times and Gazette,' January 6, 1872. In the first line of the report of the autopsy, *for* "left" *read* "right."

While awaiting further post-mortem proof of this hypothesis, we may perhaps look for a certain amount of collateral evidence from cases of cerebral abscess resulting from ear-disease. We have here gross disease of the brain, connected in all probability with the ear-disease by disease of the veins; and if, as Dr. Jackson puts it elsewhere,[1] minute changes in venous tracts may set up the cerebral changes which result in epilepsy, we might fairly expect that these minute changes would sometimes precede the greater; in other words, that epilepsy would form a point in the clinical history of patients dying with cerebral abscess in the course of an otorrhœa. Looking over the cases of cerebral abscess given by Gull and Sutton,[2] Ogle,[3] Jackson and Hutchinson,[4] I find two cases in which there was a fit before the acute symptoms began. In the first[5] there was "a convulsive fit apparently epileptic in nature" *six weeks* before admission to the hospital; the second fit occurred a week after the first, leaving permanent symptoms such as ptosis amentia, and the like. In the second case[6] the patient had had a fit *six months* before admission, leaving only pain in the forehead and a certain amount of mental affection. The second fit occurred two days after admission, and was followed by acute symptoms. It may be, however, that in these cases the first fit was caused by the presence of the abscess, as it is difficult to fix a date for its first formation, and in any case one fit does not make an epilepsy. But a third case, recorded by Jackson and Hutchinson,[7] is more to the point:—A young man had had discharge from the ears since 3 years of age, after scarlet fever. About three years before his death he fell into the Thames and was much frightened — the discharge had previously stopped. Epileptic fits followed, and continued for two years. Then pain in the right ear, offensive discharge, and death from abscess of the cerebellum. This is a clear case of chronic epilepsy occurring in a patient who finally died of abscess in the brain. Was the epilepsy, like the abscess, connected with the ear-disease?

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[1] 'Gulstonian Lectures,' 1869.
[2] Reynolds's 'System of Medicine.'
[3] 'British and Foreign Medico-Chirurg. Review,' No. 70.
[4] 'Medical Times and Gazette,' 1861.
[5] No. 99, Ogle; No. 61, Gull and Sutton.
[6] No. 104, Ogle; No. 65, Gull and Sutton.
[7] 'Medical Times,' 1861, p. 196.

Kopp and Schwartze uphold the view that epilepsy, when caused by ear-disease, is due to reflected irritation. They give two cases, the first of which is certainly striking:[1] A man, age 21, whose mother had had otorrhœa and convulsive attacks of uncertain nature, had himself ear-disease from 3 years of age, after scarlet fever. Discharge from the left ear, and attacks of earache continued on and off till the age of 15, then stopped for two years. At the age of 17, after exercise at a gymnasium, there came on violent pain in the ear, with tenderness of the mastoid process. At the same time epileptic fits began, of the following character: sense of oppression for some hours, then cold sweating; then pain in the head, gradually localising itself behind the affected ear, giddiness, spasm of the left side of the face (?), loss of consciousness (usually), and on recovery, vertigo, semi-paralysis, coldness of the extremities. These attacks were at first benefited by treatment, but became in four years' time very frequent (daily or oftener) and prolonged. On admission, there was paresis in the district of the left facial, tenderness and redness of the left mastoid process, fetid pus in that meatus, a perforation of the membrane with granulations projecting through it, and the Eustachian tube was blocked. Schwartze trephined the mastoid, and by degrees removed the pus, and opened up the Eustachian tube. A violent epileptic fit occurred just when the first incision was being made, but after this they stopped, and did not recur. From the prompt effect of the operation, Schwartze argues that the fits were reflex in character, and not due to secondary cerebral disease. In favour of this view, he urges also the cases in which epilepsy is said to have been caused by foreign bodies in the external meatus, and to have ceased with their removal. This theory, it will be noted, opens up a wider field than is covered by suppurative ear-disease only; for symptoms indicative of nervous irritation, e.g. tinnitus, vertigo, &c., occur perhaps with even greater frequency in non-suppurative middle-ear disease—fibrosis, sclerosis as it may be termed—than in purulent catarrh.

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[1] 'Archiv für Ohrenheilkunde,' Bd. V. p. 282.

I shall now quote some statistics which I have collected on the subject. They are not yet, I fear, sufficiently numerous, but I shall hope to remedy this in the future. I examined the hearing power of 100 epileptics at Queen Square Hospital, and endeavoured to ascertain the nature of the aural disease, when any was indicated by defect of hearing. (Note B.) There was a total of 17 cases of suppurative disease, past or present. Another series of 100 epileptics, whom I merely questioned as to otorrhœa, gave 14 as the number who had or had had it; this made 31 in the 200, or 15·5 per cent. This proportion might seem large enough to justify us in concluding that the two facts were connected, but to do so would be to leave out of sight the very great frequency of ear-disease among hospital out-patients. For on starting a counter-series in an out-patient room not devoted to nervous diseases (Victoria Park Chest Hospital), I found that out of 200 patients 29 had had otorrhœa, making 14·5 per cent. In this counter-series I found only one case of chronic epilepsy (and in that case there was probably aural disease); a second had been discharged from the army for "faints," possibly epileptic, this patient had otorrhœa; a third who had also had otorrhœa, had had two fits under somewhat peculiar circumstances, which I shall mention presently.

From these two series, then, no particular conclusion can be drawn, unless it be that otorrhœa is extremely common among hospital patients, and epilepsy very much less common. But a third set of inquiries gave more definite results. I inquired as to the history of "fits" from 100 patients with suppurative disease of the middle ear. These were patients under Mr. Cumberbatch at St. Bartholomew's; or Dr. Urban Pritchard at the Royal Ear Hospital, so that the diagnosis of the ear-disease rests on the best authority. Neglecting such things as "faints," hysteria, and the like, I found that seven out of the 100 had had bonâ fide epileptic fits. This is a very large proportion; for Niemeyer[1] gives the absolute frequency of epilepsy as six cases in every thousand persons; and Russel-Reynolds[2] maintains that this estimate is far too high. If then further inquiry should verify the figures which I have obtained, it would appear that epilepsy is more than usually frequent among patients with suppurative disease of the middle ear.

To turn to the consideration of particular cases. It is difficult from most out-patients to get precise accounts of their complaints, but I give the following facts as being possibly of some significance. One of my epileptic patients certainly improved when the condition of the ears was treated; he had been previously on bromide of potassium without very marked improvement. The parents of another (a boy with profuse and fetid discharge) said that the fits were always worse when the discharge was bad. A man, under treatment by Dr. Pritchard for polypus auris, had previously had fits, which he said occurred whenever the discharge stopped (when it became pent up?). A young woman had had epilepsy for eighteen months, and discharge from the left ear during the same time; during the three weeks preceding her first attendance both the epilepsy and the otorrhœa had been much worse than before. Her fits were preceded by rotatory movements. These accounts seem to point to some connection between the epilepsy and the ear-disease. As to the kind of connection, I cannot say much; but I have had three cases in which there was otorrhœa on one side only, with convulsions limited to, or principally affecting the other side. These might be urged in favour of Dr. Jackson's view, for we should expect reflex irritation to give rise to movements on the same side as the irritation; whereas cerebral disturbance from thrombosis of veins in connection with the affected ear would be manifested in the limbs of the other side.

Upon the other (reflex) theory we might justly expect that the fits would occur in close connection with some of the nervous phenomena, e.g. pain, tinnitus, vertigo, which are apt to occur in ear-disease. But here comes the difficulty, that many of these very phenomena are met with (as I have endeavoured to show) in epilepsy uncomplicated with ear-disease. Hence, we might feel unable to say whether the vertigo (for instance) was aural, or epileptic, or both. Perhaps we may be sometimes able to make out a transition from the one to the other: as a possible illustration I submit this case.

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[1] 'Text-book of Practical Medicine,' *s. v.* "Epilepsy."

[2] System of Medicine, *s. v.* "Epilepsy."

A girl, æt. 18, attending at Victoria Park Hospital for some trivial chest affection, had had earache and discharge from the right ear from babyhood. She had fits when teething, and off and on since childhood so-called "bilious attacks," consisting of pain in the head and vomiting, and beginning usually with vertigo, the patient feeling as if things were turning round and round, and as if she herself were going to pitch on to her head. Such attacks might well be due to the ear-disease. But once, while jumping with other children (being then 9 years old and in perfect health), a violent retching came on, resembling (as her mother, who witnessed it, told me) one of the "bilious attacks;" but, instead of that, she became unconscious, convulsions set in and lasted the whole day. The next day she was quite well. A similar attack came on three months later, this time while she was swinging.

The suggestion is, that in this case the equilibrium apparatus was enfeebled by the ear-disease, as may be inferred from the habitual attacks of vertigo; and that, under circumstances particularly liable to upset the equilibrium (jumping, swinging), the ordinary attack of vertigo was replaced by an attack of convulsions. It may be noted that in the case quoted from Schwartze the first fit occurred under similar circumstances, viz. after exercise at a gymnasium.

To conclude, I think that further inquiries are necessary as to the production of epilepsy by ear-disease. It is important to determine the existence of this, as indeed of any other cause of the disease. For the best means we have of checking the fits, viz. bromide of potassium, often fails altogether to prevent their recurrence when the drug is discontinued. This we might hope to do if we could act on some causal indication. It is also important, assuming the existence of such a cause, to know its modus operandi. For if it act by nervous irritation, then removal of the source of irritation, i.e. treatment of the ear-disease, must be our chief object; if by the indirect production of cerebral disease, then the epilepsy has to be treated per se, while treatment of the ear is required as a prophylactic, either against extension of the epilepsy in patients already epileptic, or against the production of it in those predisposed to become so. But in any case we must treat the ear-disease.

NOTE A.

The leading symptoms in Menière's original cases (which will be found in the 'Gazette Médicale de Paris,' 1861, and in Knapp's article in the 'Archives of Otology and Ophthalmology,' vol. ii. No. 1, p. 229) are as follows:—

Case I.—Tinnitus, 20 years, gradual impairment of hearing. Then violent attack of sneezing, followed by staggering and inability to walk in straight line. Next day, walking more difficult, involuntary motions to left, vomiting. Forty-two days later, vertigo, vomiting, convulsions of face, contortions of left side of body, followed by incomplete and transient paralysis. Hearing became worse than before.

Case II.—Tinnitus following intermittent fever. Afterwards, attacks of vertigo and vomiting: patient once fell down in them. These lasted a year; in two years complete deafness. Examination of ears negative.

Case III.—Sudden fall, pallor, perspiration, vomiting, momentary loss of consciousness, then vertigo. Loud tinnitus, deafness, dating from the attack. Several less violent attacks, leaving patient with tinnitus, increasing deafness and some constant loss of equilibrium. Examination of ears negative.

Case IV.—Sudden fall, loss of consciousness, pallor, perspiration: persistent vomiting and vertigo. Final results: uncertainty of gait, loud tinnitus, increasing deafness. Hearing had been good before.

Case V.—Frequent and sudden attacks of giddiness, nausea, vomiting: resulting in impairment of hearing, tinnitus, staggering gait. Examination of ears negative.

Case VI.—Frequent attacks of vertigo, sudden falling, loss of consciousness. Tinnitus and deafness set in and increased gradually. Attacks subsided in a year, leaving complete deafness only.

CASE VII.—"Coup de sang" in left ear, with faintness, giddiness, noises. Next night, sudden, violent and persistent vomiting, vertigo as if in a storm at sea. Deafness of left ear. Final results: uncertain gait, tendency to vertigo, deafness of left ear. Examination and treatment of ears negative.

CASE VIII.—Slight vertigo, dimness of vision, nausea, and sometimes falling, with transient loss of consciousness. Twenty such attacks in a year; then nothing but deafness. Examination of ears negative.

CASE IX.—Dimness of vision, wavering of objects before eyes, then vertigo. Vertigo recurred on sitting up during two months. Consequences; tinnitus, progressive deafness, loss of equilibrium. Examination of ears negative.

CASE X.—A "cold" caught during menstruation. Sudden deafness, persistent vertigo and vomiting. Death on fifth day. Nervous centres healthy: reddish plastic exudation in semicircular canals, traces of it in vestibule, cochlea healthy.

Menière remarks as regards the differentiation from cerebral disease:—

"Les vertiges, les étourdissements survenant tout à coup et donnant lieu à un état syncopal, à des nausées et à des vomissements, n'appartiennent pas exclusivement à une lésion de l'oreille interne: il peut se faire que ces désordres symptomatiques dépendent de certaines affections cérébrales, d'un état congestif des méninges, de quelque lésion du cervelet et de ses dépendances; mais nous croyons que, quand les symptômes s'accompagnent de bourdonnements des oreilles, de bruits continus, variables, et surtout quand on voit bientôt survenir une diminution notable de l'ouïe, alors le mal a son siège dans le labyrinthe, et plus particulièrement dans les canaux demi-circulaires."

And of the general characters of the disease he gives the following summary:—

"1. Un appareil auditif, jusque-là parfaitement sain, peut devenir tout à coup le siège de troubles fonctionnels consistant en bruits de nature variable, continus ou intermittents, et ces bruits s'accompagnent bientôt d'une diminution plus ou moins grande de l'audition.

"2. Ces troubles fonctionnels, ayant leur siège dans l'appareil auditif interne, peuvent donner lieu à des accidents réputés cérébraux, tels que vertiges, étourdissements, marche incertaine, tournoiement et chute, et, de plus, ils sont accompagnés de nausées, de vomissements et d'un état syncopal.

"3. Les accidents qui ont la forme intermittente ne tardent pas à être suivis de surdité de plus en plus grave, et souvent l'ouïe est subitement et complétement abolie.

"4. Tout porte à croire que la lésion matérielle qui est cause de ces troubles fonctionnels réside dans les canaux demi-circulaires."

NOTE B.

Analysis of 100 unselected cases of epilepsy, with respect to the aural symptoms presented by them: to show the frequency with which such symptoms occurred in those whose ears were sound or diseased :—

Fifty-four cases were normal as to hearing power; and of these :—

Noise in the ear, as aura	occurred in	4	cases =	8 per cent. (or just under.)
Do. independently of the fit . . .	„	2	„	4 per cent.
Pain in the ear	„	3	„	6 „
Vertigo, definite in character . .	„	11	„	22 „
Do. vague (staggering, &c.) . . .	„	7	„	14 „
Vomiting or nausea	„	12	„	24 „
Well-marked rotation	„	2	„	4 „

(The vomiting and vertigo were associated in 5 cases, in one of which also the rotatory movements were present.)

In 21 cases there was slight deafness; the cause of which appeared to be :—

In 6 cases, wax.
„ 3 „ slight thickening or depression of the membrane.
„ 6 „ doubtful.
„ 1 „ central mischief, post-epileptic deafness.
„ 5 „ previous catarrh, with history of discharge.

Out of these 21 :—

Noise in the ear, as aura. . . .	occurred in	2	cases =	10 per cent.
Do. of uncertain nature	„	1	„	5 „
Pain in the ear, after the fits . .	„	1	„	5 „
Do. in connection with discharge or of doubtful nature	„	2	„	10 „
Vertigo, definite in character . .	„	7	„	35 „
Do. vague	„	4	„	20 „
Vomiting	„	1	„	5 „
Rotatory movement (with vertigo).	„	1	„	5 „
Do. (with vomiting)	„	1	„	5 „
Deafness, increased by fits . . .	„	2	„	10 „

In 25 cases there was well-marked deafness; the cause of which appeared to be :—

In 12 cases, suppurative middle-ear disease (present or past).
„ 6 „ chronic thickening or depression of membranes.
„ 4 „ wax.[1]
„ 3 „ of doubtful nature.

Out of these 25 :—

Noise in the ear, as aura	occurred in	1	case =	4	per cent.
Do. independently of the fits.. . .	„	2	„	8	„
[Pain in the ear occurred in connection with suppuration.]					
Vertigo, definite in character. . .	„	6	„	24	„
Do. vague	„	6	„	24	„
Vomiting	„	3	„	12	„
Rotatory movements	„	1	„	4	

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[1] One of these cases subsequently developed paralytic symptoms, sufficient to take it out of the category of simple epilepsy.

THE INFLUENCE OF HEREDITARY SYPHILIS IN THE PRODUCTION OF IDIOCY OR DEMENTIA.

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AMONGST the many causes of idiocy enumerated by writers on insanity, the influence of syphilis has not yet, I venture to think, received the attention due to its importance. The à priori probability of idiocy being a frequent result of inherited syphilis is here and there referred to, but those who have investigated the subject have almost invariably come to the conclusion that the connexion between the two must be a rare one. At the outset it is difficult to receive such a view of the matter without surprise, when it is remembered (1) that cases of insanity from acquired syphilis are by no means uncommon, and (2) that all the morbid changes found in the skulls or in the brains of such cases are also met with in the subjects of hereditary syphilis: indeed, the number of recorded post-mortems of gross brain lesions in hereditary syphilis stands in marked contrast to the very few observations on mental disturbance in this disease. But it seems improbable, to say the least, that thickening of skull, or of brain membrane, or of brain arteries, will be less likely to damage the mental faculties by their effects on a sensitive developing brain than by their effects on a fully grown one; and just as in epilepsy the tendency to mental failure is greatest in the cases which begin in childhood, so we should naturally expect that a similar relationship would hold in syphilis. But the study of inherited syphilitic nervous diseases being still in its infancy, and opportunities for making post-mortems on idiots not too numerous, it is impossible at present to form a definite idea as to the frequency of syphilitic idiocy. The cases related further on afford, at least, definite proof of its existence; before describing them, some of the chief allusions to the subject found scattered through medical literature may here be noticed.

Mr. Critchett[1] mentions a case of idiocy in a boy, æt. 9 years, who had double interstitial keratitis, pegged and notched teeth, and perforation of the soft palate. A history of congenital syphilis. He did not walk till 4 years old, and had never learned to talk. Hearing good; a ravenous appetite. At times very violent. Mr. Critchett remarks: "There does not appear to be any reason for supposing that actual idiocy is at all a common consequence of inherited syphilis. About three years ago, we looked through the patients at the Redhill Asylum without finding a single one whose teeth were characteristic, and Dr. Down informs us that he has recently made another inspection with a like result. Impairment of intellect is very common among the syphilitic, and is usually accompanied by a mis-shaped protuberant forehead; but the condition, according to our observation, rarely approaches an entire loss of intellectual power. The case is the only instance of the kind which has yet come under our notice."

Mr. Hutchinson,[2] out of a total of about 170 cases of hereditary syphilis, notes an idiotic condition in only three of them.—1. Female, 8 years, keratitis of right eye, one of upper incisors characteristic, fissures at the angles of the mouth. She is hydrocephalic and idiotic; she had fits when a week old, and her head rapidly increased (p. 89).

2. Half-idiotic boy, typical teeth, marked syphilitic physiognomy, head large and mis-shapen. Subject to convulsive fits while teething. Tall and well grown; almost total blindness and white atrophy of the optic nerves. "His mother considered him clever, but he had never learnt to read, and her surgeon had frequently urged upon her that the boy should not be allowed to work his head" (p. 164).

3. Female, 10 years, typical physiognomy and teeth, total blindness from white atrophy of optic nerves, choroidal changes.

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[1] 'Medical Times,' vol. i., 1860, p. 575.

[2] 'Diseases of the Eye and Ear consequent on Inherited Syph.' London, 1863.

"Evidently partially idiotic. She was exceedingly fretful and restless, frequently crying or laughing, often dancing about the room. She answered questions in a wild manner, wandering off to some other subject directly. Her mother assured us that these symptoms had come on within a few weeks, but of this I felt much doubt." She had excellent sight till about 7 years of age, and was precociously intelligent (p. 169).

Mr. Hutchinson also "examined in company with the writer the teeth of the idiots at the Earlswood Asylum, but did not find the specific character in any considerable number of cases."[1]

Zambaco[2] relates the case of a man, æt. 26 years, who for about a year had been subject to sudden and temporary losses of consciousness, which came on when quite still and could be avoided by walking about; he had paresis of lower limbs, frequent pains in head and limbs, and *loss of memory*. A strong family history of syphilis. Patient had various eruptions in infancy, and ulceration of the palate when 12 years old, which Nelaton pronounced syphilitic. A careful investigation excluded acquired syphilis. Zambaco thinks that the presence of an exostosis would best explain the various phenomena.

Lancereaux[3] records the case of a child, æt. 12 years, who was microcephalic, a complete idiot, and had had epileptiform attacks since the age of two. Necrosis of the tibia for several months. Father syphilitic. Mother had had four miscarriages and four living children, of whom the patient was the only survivor.

Griesinger,[4] amongst the diseases of childhood, mentions syphilis as a cause of idiocy, and refers to two cases: one described by Erlenmeier, of an idiotic child, where the cranium presented numerous exostoses, and who recovered under the use of iodide of potassium; another by Guislain, "where a child, begotten of a powerful man while undergoing a course of mercury for syphilis, whose former and subsequent children were perfectly healthy, was idiotic."

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[1] 'Manual of Psych. Med.' Bucknill and Tuke. London, 1879. Footnote to p. 161.

[2] 'Affect. Nerv. Syph.' p. 207. Paris, 1862.

[3] 'Traité de la Syph.,' p. 442. Paris, 1873.

[4] 'Mental Path. and Therap.' London, 1867.

Dr. E. Mendel[1] reports very fully by far the most important case that I have been able to find, and he seems to be the only writer who has considered the subject worthy of a separate paper. The following is an abstract of the report:—

Agnes B., born of a family whose members had never suffered from mental or nervous affections. At the age of 2 months an eruption broke out and the child began to waste; at 7 months the nurse noticed the rash on the back, on the hands and feet, and ulcers at the anus; the hair of the head, eyebrows and eyelids fell off, and a doctor said the child was syphilitic, he found the mother had secondary symptoms. After taking white powders the child got healthy; when 1½ years old, learned to walk; when 3 years old, an external squint of the right eye came on suddenly and the right pupil dilated. At 7 years of age went to school, and learnt figures and French. Between 7 and 9 years, slight inflammation of eyes and glands of neck. At 9 years the first signs of mental failure, she came to a stand-still in school work. At 11 years choreic movements, which disappeared in a few months. Her intellect remained as at 9 years, but her temper became more and more excitable. At the age of 15 years she had maniacal attacks, with delirium and hallucinations; these were soon followed by a complete state of apathetic imbecility, in which condition Dr. Mendel first saw her. For her age, 16½ years, body well developed; functions, with exception of menstruation, normal. Right external strabismus excessive, right pupil dilated and inactive to light. Gait uncertain, tottering, with a forward inclination of the upper part of the body. Strong trembling movements of the tongue, hands and arms. Expression quite stupid; when asked questions, either does not answer at all or stammers out some unintelligible sounds. Sits or stands the whole day without doing anything. Urine and fæces passed anywhere. Three months later a glandular swelling formed near the right angle of the lower jaw, which suppurated in about three months; after the abscess was opened, her mental condition seemed to improve a little. But her old condition soon returned again; she wasted more and more, standing or walking became more difficult. Finally she took to her bed, convulsions set in, affecting chiefly the right side, and she died aged 18 years.

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[1] "Ueber Hereditäre Syph. in ihrer Einwirkung auf Entwickelung von Geistes. Krankheiten." 'Archiv für Psychiatrie,' Berlin, 1868–1869, S. 308.

Autopsy.—Skull cap universally and equally thickened, but felt light owing to very vascular diploe; it adhered to the dura mater at a spot near the hinder end of the sagittal suture on the left parietal, and here was a depression of the internal table. At the base of the cranium, half an inch outwards from the left edge of the foramen magnum, was a conical exostosis about 8 lines long. Arteries healthy. Dura mater not much thickened, but at the place where it was fast to the bone it was also adherent to the brain substance. The arachnoid, (pia mater?) especially on the convexity, was turbid and thickened, it could be easily detached from the convolutions, except at the spot mentioned above. Brain anæmic, œdematous and softened. The left lateral ventricle was much dilated; the posterior and inferior horns presented a cavity about the size of a walnut, which contained 1½ oz. of a yellowish clear fluid: the dilatation was so great that the hinder end of the left hemisphere was extremely thin. The brain ganglia were flattened; the ependyma looked granular. The right ventricle was also dilated and contained a little serous fluid. Pons somewhat soft. The cerebellum was of good consistence, over the position of the exostosis the left hemisphere was somewhat flattened. The fourth ventricle was a little widened.

Dr. Mendel remarks that the wide-spread meningitis, the adhesion of membranes, and the exostosis, together with the whole course of the disease, all point to its syphilitic origin. He thinks that a specific periostitis of the skull-bones was started soon after the disappearance of the syphilitic eruption, and he attributes the various motor and psychical phenomena to the gradual extension of this periostitis, and to the disease of the membranes and of the ventricles. Dr. Mendel also refers to cases reported by Rosen, Engelberg, Howitz, Haase, Bärensprung, Virchow, of young children of syphilitic parentage who died of hydrocephalus. Other observers, such as De

Méric,[1] Lancereaux,[2] also admit the influence of syphilis in the production of hydrocephalus.

Virchow[3] says that the causes of congenital encephalitis and myelitis are the acute exanthemata[4] and syphilis; the disease is characterised by a fatty metamorphosis of the cells of the neuroglia. Virchow thinks that when the disease does not kill, it may often lead to paralysis in children and to many cases of idiocy.

Dr. Hughlings-Jackson,[5] in his most suggestive paper on "nervous symptoms in cases of congenital syphilis," relates the following very interesting example of syphilitic dementia:—Samuel D., 15 years, pegged teeth, remains of choroiditis in the left eye, and the left disc atrophic: family history of syphilis unmistakeable. *Normal mental history:* As a child he was rather precocious, talked early, learned to read and write early, was fond of reading, wrote good letters, had an excellent memory, he was good tempered, was fond of music and very religious. He never did odd things nor behaved strangely. *Abnormal mental condition:* "He looked listless and sullen, and was evidently fatuous." "Some months before his actual mental failure a ship in the yard where he was at work caught fire, and this seems to have affected him a great deal." "He became very much addicted to masturbation, then his memory began to fail; he got dull and stupid, took no interest in things, but would sit by the fire and fall asleep. He became very sleepy, and would fall asleep under any circumstances." For several months he had been in the habit of bringing home and hiding things that did not belong to him in the workshop. His temper became very violent. He died two years later from some fever. "For the last six months of his life he stammered." "He never lost power in either hand, never had a fit. A fortnight before his death he lost his speech altogether, but the mother is sure from his gestures that he retained his consciousness." No autopsy. Dr. Jackson says that the choroiditis suggested a "pia matritis," and thinks that "there was local syphilitic disease, followed by general atrophy of the hemispheres." "My own speculation is that the boy had by inheritance an imperfect brain, and that any sort of disease, syphilitic or otherwise, would have led easily to *loss* of the higher intellectual and emotional faculties, and indirectly from this 'loss of control,' or lack of inhibition to increased play of the lower faculties." "The boy's father died insane, and this fact supports the inference just drawn. There were two factors, an imperfect brain by inheritance and disease."

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[1] Lettsomian Lectures; the 'Lancet,' 1858.

[2] *Loc. cit.* p. 432.

[3] 'Archiv,' Bd. xxxviii. p. 129.

[4] Dr. A. Rosenthal, in a paper, "über die anatom. Veränderungen im Gehirn bei infektiösen Krankheiten" (Med. Centralbl. xix. 20, 1811) describes changes in the intima of the vessels of the cortex, in the large pyramidal cells constituting Meynert's 3rd layer, in the medulla oblongata, and in the 4th vent. He considers the chief changes to be parenchymatous, and that they afford an explanation for the brain symptoms in the course of the infectious disease and for those occurring subsequently.

[5] 'Journal of Mental Science,' 1875.

In the "London Hospital Reports," vol. i., 1864, Dr. Jackson gives the following particulars of a syphilitic family. The eldest, a girl of 18, had good teeth, but remains of old iritis, and scars at the angles of the mouth. A girl of 15, with typical teeth, partial blindness from choroiditis, and paresis of the right limbs. A girl aged 12, who had specific teeth and choroiditis. A boy aged 8, paraplegic, partly *idiotic*, and who had had several fits: he was quite blind, both optic discs were dirty white, margins obscure, vessels small, and fundus hazy.

Dr. George Thompson[1] alludes to an idiotic girl in the Bristol Asylum, "whose arrested development seems to be due to hereditary syphilis, as she is blind through interstitial keratitis, and has the well-known notched teeth and imperfectly developed alveolar process described by Hutchinson: when first admitted she was the subject of paroxysms of excitement, lasting several days and nights at a time." These attacks were much benefited by 20 minim doses of tinc. bellad.

Ireland[2] says that idiocy "does not seem to be a frequent consequence of hereditary syphilis, though I have met with a few apparent instances of the kind."

Maudsley,[3] in speaking of melancholia in children, says:— "The nervous or psychical *tone* is radically infected with some vice of constitution, so that every natural impression, instead of being pleasing, is painful. The cause of the defect, in some instances, is inherited syphilis; at any rate, beneficial results follow the treatment for hereditary syphilis."

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[1] 'Journal of Mental Science,' 1874. [2] 'Idiocy and Imbecility.' London, 1877.
[3] 'The Pathology of Mind.' London, 1879.

Echeverria[1] remarks on the rarity of epilepsy from inherited syphilis; he briefly reports eight instances, and of these, six were more or less demented.

To sum up the information derived from the preceding observations, there is (1) distinct proof afforded by the post-mortem examination of Dr. Mendel's case that mental failure in childhood may be the result of lesions produced by hereditary syphilis, and (2) there is fair evidence that hydrocephalus, and other fœtal brain changes, sometimes result from syphilis, and may lead on to idiocy; but we know nothing of syphilitic idiocy simply from inherited weakness of nerve elements, apart from demonstrable changes, which are strictly extra-neural.

The following cases are related as a further contribution to the pathology of hereditary syphilitic dementia. For the use of the first I am indebted to Dr. Henry Humphreys.

Case I.—Mary G., æt. 11½ years, was admitted into the Children's Hospital, Pendlebury, November 25th, and died on December 18th, 1878.

Family history.—Father has had sore throat, but is in general healthy. There is consumption on the mother's side. They had fourteen children, of whom the patient is the only survivor; the deaths all occurred before the age of 13 weeks; all the infants had snuffles and convulsions.

The patient is said to have been fairly healthy until she was three years old, then she had a fit, in which the right side worked, she did not lose her senses; the next day she could move the right hand, but dragged the right foot. Three weeks afterwards she had a second fit, and then others followed at varying intervals. At times the fits lasted the whole day, only the right side moving. She has had no fits during the last 18 months. She became unable to walk about 4½ years ago. Has not been able to feed herself for 4 years: 2½ years ago she practically lost her speech, and now she can only say

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[1] 'Journal of Mental Science.' London, 1881.

"cockles," and one or two other odd words. On admission, completely idiotic, much emaciation, skin yellowish. She lies crouched up in bed; face generally expressionless, at times assuming an idiotic smile. Makes a few inarticulate noises at times, but does not speak. Follows objects moved before her with her eyes. Everything given her she puts into her mouth. Catches hold of objects in a feeble, awkward way, being unable to extend the wrists. Is not able to stand. The middle incisors of upper jaw are typically notched and pegged. The axes of the eyeballs appear to be slightly divergent, but no loss of any particular movement can be detected. The optic discs are chalky white, the vessels threadlike, the choroidal spaces are very apparent: in the left eye blotches of black pigment are seen in the choroid, this appearance is not noticeable in the right eye.

December 10th.—Had two convulsions last night, and one this morning, in which the right limbs moved. The right arm is now drawn up. In a fourth convulsion the eyes were drawn upwards, and to the right, the left naso-labial fold was strongly marked, and the left angle of the mouth and the left eyebrow twitched. Autopsy December 19th. Weight 35¾ lbs. Much emaciation. Scalp very adherent; the occipital bone projects considerably above the level of the parietals. Skull-cap slightly thickened, the bone dense. Longitudinal sinus empty. Much fluid at base of brain. Attached to the internal surface of the dura mater on the right side was a thick fleshy vascular false membrane, which could be stripped without difficulty from the dura mater; it was best marked over the vertex. There was no false membrane on the external surface of the dura mater. On the left side there was a similar condition, only to a much less marked degree, the false membrane here was thin and shreddy. The pia mater was everywhere thick and tough, and in many places presented a milky turbidity; on stripping it away from the brain it left a smooth surface. The hemispheres were non-symmetrical, for on the left side the temporo-sphenoidal lobe was much shrunken, and was smaller than the corresponding portion of the other side. On both sides the sulci were remarkably deep and broad, and the convolutions, as a rule, *exceedingly narrow and wasted;* the first and second frontal convolutions, however, were of fairly good breadth. Optic nerves greatly wasted, optic tracts still more wasted. Brain substance *firm*, presenting no signs of coarse disease. The cerebellum presented appearances similar to those in the cerebrum. Weight of brain 28¼ oz. The middle and anterior cerebral arteries stood out like cords, and their surface had a milky appearance, their walls were decidedly thickened. The spinal cord appeared healthy to the naked eye; no thickening of membranes noticed.

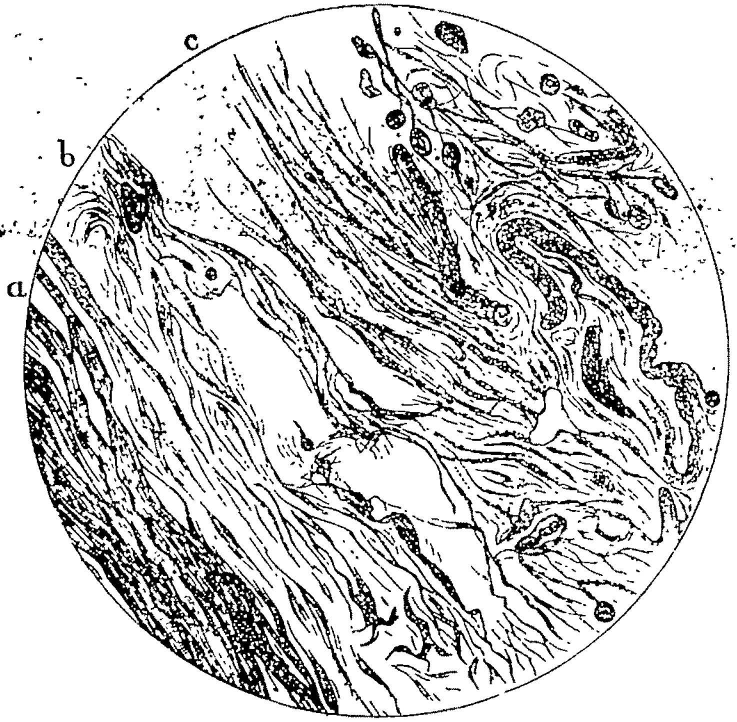
Skull.—The base of the skull presented a most remarkable overgrowth of hard dense bone; the lesser wings of the sphenoid bone and the petrous bones, especially the left one, were exceedingly thick; the left squamous bone presented such a large mass of new bone that the capacity of the corresponding middle fossa of the skull was thereby greatly reduced; the groove of the middle meningeal artery was very large. No nodes on the external surface of the skull.

Lungs.—At the apex of the lower lobe of the left lung there was a nodule of new growth, about the size of a hazel nut; it was encapsuled, and was traversed by fibrous bands; in consistence it was fleshy. Around it were nodules of greyish-green exudation, mostly of the size of pin-heads. There was a similar nodule in the upper lobe of the left lung, and in one of the lobes of the right lung.

The *liver* weighed 19½ oz.; capsule presented some milky tracts; and a few pale greyish spots, the size of pin-heads, were found on section to dip downwards a little way into the liver substance. The tissue of the liver was tough. The *spleen* weighed 2 oz.; the capsule was considerably thickened, and of a yellowish-grey colour; tissue pale on section. The *heart* and *kidneys* appeared healthy.

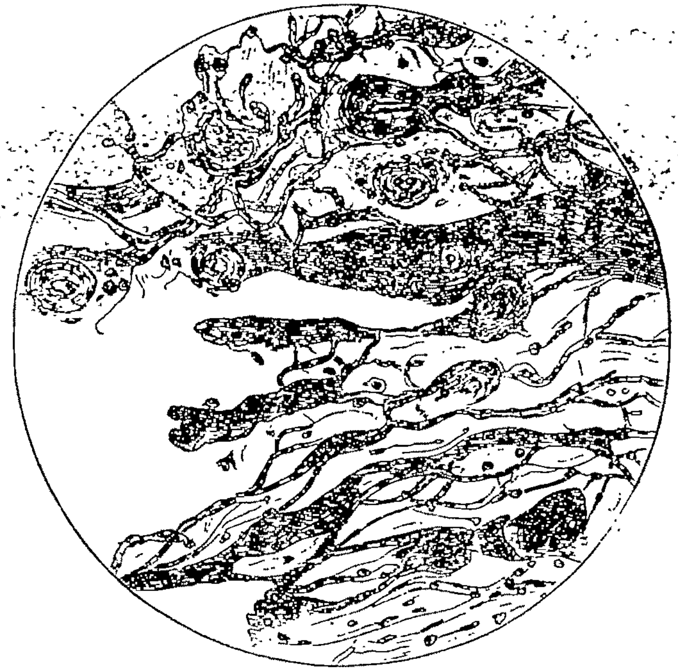
No nodes were felt on the long bones. A microscopical examination was made of the middle cerebral arteries, of the false membrane attached to the dura mater, and of the nodule in the lungs. The arteries show the condition known as that of syphilitic arteritis, the chief thickening being between the endothelium and the fenestrated membrane. The false membrane is made up of parallel layers of vessels with a little connective tissue between them; the vessels, which are chiefly capillaries, have remarkably thick walls, which are studded with numerous nuclei. The changes are well shown in the accompanying woodcuts from drawings by Dr. C. S. Watson, —in the first, drawn under a low power, *a* represents the dura mater, *b* and *c* the two layers of the false membrane; the thickened vessels in *c* are seen more highly magnified in the second figure. The nodule in the lung presents the appearance of a syphilitic gumma in that locality, as described by Dr. Goodhart in the 28th volume of the Path. Trans.

Fig. 1.



This case illustrates, in the most striking manner, the various syphilitic lesions that are able to produce atrophy of brain; the thickened bone, the narrowed arteries, and the diffuse meningitis are each individually capable of impeding brain growth. It is to be noted that although the convulsions were on the same side as the false membrane lying on the convolutions, there was no evidence of any active process going on in the membrane; it is therefore probable, that the right-sided epileptiform seizures are best referred to sudden and transitory interferences of the circulation of blood in the left cortical motor region, as a result of endoarteritis. The only recorded case of pachymeningitis interna occurring in hereditary syphilis is related by Heubner in the last volume of Virchow's 'Archiv.'

Fig. 2.



Case II.—Mary A., healthy as a baby till vaccinated, when she became covered with brown spots, also snuffled in nose. She talked sensibly, and played with other children, and had good memory *till* 8 *years old;* used to learn hymns and sing them, and was fond of music. When æt. 12 years, had a fit in which the right side was chiefly drawn up; the parents state that it was not "a proper convulsive fit: she lay for about 24 hours partly unconscious, and drew herself up." "After the fit the right foot trailed in walking, and then the *right* side became paralysed." She often had pain in the head. She had two more similar fits before her death; after the third fit she never spoke, that is, about 9 months before she died. The mother has had 9 children; 2 are now living and fairly healthy; 6 died in infancy of "convulsions and teething"; all her babies snuffled and had brown spots. No family history of insanity or of nervous diseases. I saw the patient for the first time a year ago, then 14½ years old; she was lying in bed, and was quite demented; there was some paresis of *right* limbs, no rigidity. The upper central incisors were typically pegged, but not notched. Old scars at the angles of the mouth, and a characteristic forehead and physiognomy. I only saw the child once again before death, in the Crumpsall workhouse hospital, through the kindness of Dr. Bailey, last August. She was then lying crouched up in bed, her arms and legs rigidly flexed. Emaciation extreme. The last stage of dementia. During the 6 months at Crumpsall, she had been subject to attacks of screaming, had not had a convulsion, had never spoken. Hearing and sight apparently unaffected. Very vicious, used to try and bite. Said to have been paralysed on the *left* side when she was admitted to hospital. She died a few weeks later, aged 15 years.

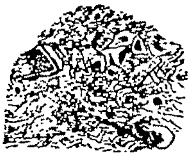
Autopsy.—The two most striking things were the excessive emaciation and the extreme muscular rigidity; the knees and elbows were so firmly flexed by the contracted tendons that they could not be straightened in the least degree. The skull-cap was not thickened. The dura mater was slightly thickened; on cutting through it, about 3 oz. of clear serous fluid escaped from the arachnoid cavity. There were some very minute granulations on the surface of the arachnoid, chiefly near the longitudinal fissure. The pia mater was thick, very opaque, and presented many milky-looking areas; the thickening of the pia mater was specially marked over the vertex, near the inner borders of the hemispheres. Nearly all the fissures were filled up with yellow thick sticky lymph, which glued together the adjacent parts of the brain. The convolutions were slightly but decidedly wasted; there was no want of symmetry between the two sides. The brain arteries were patent, firm, and the smaller branches stood out much more distinctly than normally. The brain substance was, if anything, *firmer* than natural; it was very carefully examined, but no gummata, no softenings or other local lesions were found. Cerebellum natural.

The wasting of the convolutions was not nearly so great as in the case of Dr. Humphreys, nor were the large arteries so thickened.

Numerous sections were made of pieces of the cortex, taken from the upper end of the ascending frontal and parietal convolutions, of the dura mater, of the middle and anterior cerebral arteries, and of the other organs. The changes found are briefly as follows. The thickened pia mater has a loose fibrillated structure, and contains well-formed rounded cells, which are numerous on the surface adjacent to the cortex, and here and there they can be traced extending along the sheaths of the small arteries which dip into the cortex; these and other arterioles in the pia mater and in the cortical substance present a hyaline thickening of their walls; all the small vessels are distended with blood, one or two small arteries contain an organising thrombus.

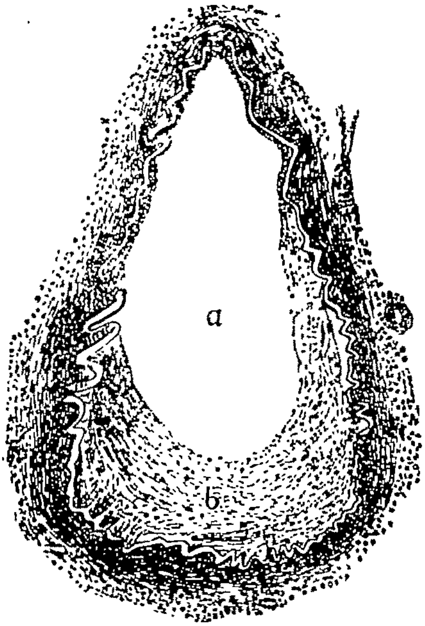
The remarkable feature of the cortex is the absence of nerve cells; in some sections of the grey matter *not a single cell* can be found, in others, a few are found; in parts of other sections the cells are fairly numerous, but still by no means so numerous in any part as in health; scarcely any of them are as large as a healthy cell, the majority of those found are not much larger than a leucocyte. The drawing from a carmine stained section shows a group of atrophied pyramidal cells with wide pericellular spaces; it will be seen that one or two of the cells are scarcely larger than the blood-corpuscles in the neighbouring vessel. Dr. Ross, who kindly looked over the sections, was also much struck with the absence of nerve cells. The place of the nerve cells is taken by a delicate fibrillated tissue, dotted over in logwood sections with small brightly-stained nuclei. The walls of the middle and anterior cerebral arteries are thickened, in both cases the two outer coats, but the anterior cerebral do not show any thickening of the intima; the

Fig. 3.



middle cerebral, however, present the characteristic subendothelial nodules first fully described by Heubner; that is, there is a growth of well-formed, round and fusiform cells between the endothelium and the fenestrated membrane; in the camera-lucida drawing the letter *a* is placed in the lumen of the artery, *b* in the middle of the thickened inner coat. A small piece only of the *spinal cord* was obtained from the upper opening of the canal; sections show sclerosis of the lateral columns, and also of the internal aspect of the anterior columns, the change being slightly more marked on one side than on the other. The *liver* was large and congested; the capsule was here and there thickened and puckered. The *spleen* was small and tough, and looked fibrous on section; the microscope showed increase of fibrous tissue. The *kidneys*, about normal size, presented remarkable groups of small bodies, about the size of miliary tubercles, on the surface of the cortex; on cutting through one of them, it was seen that they formed the tops of white streaks which reached the base

Fig. 4.



of the pyramids; it is very difficult to say whether they are gummata or tubercles: in favour of the former are, I think, the following points. They are made up of well-formed cells, round, tailed and fusiform; these cells are not collected into such definite heaps as is the case with tubercles, they also extend between the renal tubules; there is no trace of caseation.

The interesting features of the case are:—

1. The meningo-encephalitis leading not to softening but to sclerosis[1] of the cortex, with resulting atrophy and disappearance of the nerve cells, and hence dementia. 2. The bilateral descending degeneration from disease of the cortical motor areas.

Case III.—Annie L.,[2] æt. 14 years, came to see me at the Clinical Hospital last February, when the following notes were taken:—

Family history.—The mother had 10 full-time children and 2 miscarriages: (1) dead born, when mother 8 months pregnant she had brown spots all over her body, and the doctor called it the "bad disorder"; (2) died 4½ years—"would have been an idiot if lived"; (3) the patient Annie; (4) died in convulsions, æt. 6 weeks; (5) living, æt. 11 years—often complains of head—snuffled when a baby; (6) living and well; (7), (8), (9) died in infancy of chest affections; (10) living, 2 years. No mental weakness in any of relatives, no history of fits or other nervous diseases. The patient when a baby was one mass of spots, had snuffles, screamed a great deal. Subsequently pretty well, till her weak state of mind came on about 4 years ago. For the last 12 months her walking has been bad, power to hold her water has been getting less, and her hands and mouth have occasionally twitched. She is a well-nourished girl, pleasant intelligent face, well-shaped head. The upper central incisor teeth are not pegged, but there is a shallow notch in the lower edge; the teeth are far from being typically specific, but they are suggestive. There is well-marked disseminated choroiditis. Dr. Glascott, who subsequently kindly examined the fundi, said he had no doubt of the changes being syphilitic; he pointed out that they were most advanced in the right eye. The corneæ are clear. The speech is drawling; when asked her name, states it correctly; comprehension very slow. She is very nervous, easily frightened, very irritable during examination, whines a good deal. Her mother says that she is very sympathetic, cries if mother cries; "never gets excited, if plagued, cries very easily"; "starts at the least noise"; "sits and takes no notice, does not speak unless spoken to, then talks of things that happened a long time ago." She cannot read letters, she could 2 years ago, but never able to read a book. The knee jerk is exaggerated on both sides. Temperature normal. Extremities very cold. Urine free from albumen. Feeble action of the heart. The girl attended as an out-patient for about 3 months, during which time she took iodide of potassium and belladonna without any benefit. Her mother then got her into the Chorlton Union Hospital, and after a short stay there she went to the Lancaster Idiot Asylum, where, through the kindness of Dr. Shuttleworth, I saw her last November. Her condition was much the same. Dr. Taylor has lately very kindly supplied me with the following additional notes of her mental condition:—

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[1] An extremely interesting case of sclerosis of encephalon in a girl, aged 10¾ years, the subject of inherited syphilis, is reported by Dr. Samuel Gee in 'St. Bartholomew's Hospital Reports,' 1880. General convulsions with coma at 3 years, lost the use of right side suddenly at 6 years, but able to walk in three weeks. *On admission.*—Specific teeth, "*understanding not very high,*" slight paresis of right limbs, retino-choroiditis of left eye, detached retina in right eye: subsequently dropsy, albuminuria, total blindness. *Autopsy.*—Meningitis, much atrophy of left hemisphere and a little of right, convolutions *harder* than natural, some other parts of brain firmer than natural, a *remarkable absence of nerve cells* from deeper layers of cortex, and an increase of neuroglia: middle cerebral arteries thickened with gelatinous nodules.

[2] This patient was shown at a meeting of the Manchester Medical Society, March, 1882.

"She can't read, she can name a few common objects, such as a doll, a watch; she calls all coins either a 'penny' or a 'sovereign'; she has no idea how many fingers she has after she has counted them; seldom answers a question unless repeated more than once, and there is a tendency to repeat the question. She does not notice what is going on around her, and seldom speaks unless spoken to. She does not, as a rule, indicate her wants in her way. She never cries, and seems always quite happy. Feeds herself with a spoon, but is apt to use her fingers. Is very timid and nervous, never sad, nor has any vanity been noticed, is not at all vicious, readily laughs when not frightened; when frightened, muscular tremors are noticed. If left alone, she sits with her hands before her, moving her tongue from side to side of her mouth; she is pleased with a doll, and will sit rocking it quite happily. If a set of coloured discs are set before her, and she is asked the name of one of them, she either does not answer or repeats the question; if the name of a colour is suggested, as 'blue,' she says 'it's blue,' and then calls them all 'blue' indifferently. If told to match a colour she can't do it, probably because she does not understand what is wanted, for I do not think we can infer that the perception of colour is gone."

Dr. Taylor also informs me that the superficial reflexes are well marked, except the plantar, and this is less marked on the left than the right side. He also finds the knee jerk greatly exaggerated on both sides, the slightest touch producing it, and obtainable by percussion of the tibia. Ankle clonus absent. Hearing, smelling, and tasting seem normal.

The mental failure in this case, together with the choroiditis and other symptoms, suggest some thickening of the pia mater, and perhaps cortical change, and the excessive knee jerk is an ndication of possible descending sclerosis.

Dr. Orchard, of the Chorlton Hospital, kindly sent me this child for examination :—

CASE IV.—Hannah H., æt. 16 years, parents dead; father died of hemiplegia, "probably syphilitic," Dr. Orchard says. He also tells me that her brother has syphilitic teeth. She has been demented about 6 months; was deaf and had bad sight when admitted to the industrial school, about 5 years since, but was then quite sensible, and the trainer said the best help in the school (as regards manual work, for had never learnt to read). Hearing is now quite lost, and the deafness is symmetrical; there is no active ear mischief. If left alone, patient sits still and does not attempt to do anything, will not offer to get food for herself. Is bad tempered. Uses very filthy language, did not do so before her mental failure. Can distinguish between different kinds of food, and between ordinary objects. Very frightened at times, tries to catch at something in the air. Very restless at night, will jump out of bed screaming, but the nurse can soon quieten her. Staggers in walking. The upper central incisors are typically pegged and notched.

Eyes: Tag of adhesion at pupillary margin of right eye, indicating old iritis. Symmetrical disseminated choroiditis, accumulations of pigment, and patches of atrophy.

Heart and other organs seem healthy.

The signs of inherited syphilis in this case are well marked: the characteristic deafness, described by Mr. Hutchinson, preceded by 3 or 4 years the mental failure.

For the notes of the two following cases I am indebted to Dr. Henry Ashby, Physician to the Pendlebury Hospital:—

Case V.—Thomas B., æt. 8 years, admitted Oct. 16, 1882. "Snuffles" at birth and eruption on buttocks, always delicate; abscess in groin when 15 months old, which lasted 9 months. Teeth were very bad, the roof of the mouth often swollen and soft. Went to school when 4 years old, learnt his letters and pieces of poetry. His sight began to fail him after he had been at school a year. He was well in other respects. Then he became very sensitive to noises, used to put his hands to his head and complain of pain. Gradually got worse. His mind began to fail during the next 2 years (about 5–7 years old). His legs also began to fail him 2 years ago, has been quite unable to walk for 18 months. Before he went off his feet he had frequent falls, mostly backwards, sometimes forward. Parents healthy, so far as can be ascertained. A younger brother has hydrocephalus. Patient's head only closed when he was 5 years old, always had a large head.

Oct. 18*th.*—Since admission, night and day has almost continually been jabbering "ding, dong," "quack, quack;" hums to himself; never sensible for a moment. Does not sleep well at night. Takes food well. Apparently quite blind. Can't stand. Lies with knees drawn up to chin, never puts them down; can be forced down, but are then rigid, and soon are drawn up again. When extended, the thighs are adducted; knees together or crossed; feet, talipes varus; the condition of spasm varies, but is never entirely relaxed. Superficial reflexes apparently absent. No knee-jerk. Large scar about anus, the result apparently of old syphilitic ulceration. The upper teeth consist of worn-down stumps. The optic discs are chalky-white and small, edges well-defined; pupils semi-dilated and fixed. Rotary movements of the head at times. Draws up feet on pricking toes or soles.

Oct. 20th.—Some cremasteric reflex noticed to-day.

Nov. 13th.—Hearing is good.

Nov. 21st.—In same condition. Passes fæces and urine under him. Usually lies with one hand under his head. The arms seem all right.

CASE VI.—Sarah K., 9½ years; rash over body soon after birth, has always been ailing since, other children healthy. On *admission*—anæmic girl, is almost quite deaf and *wanting in intelligence;* upper central incisors peg-shaped and notched at lower edge; four lower incisors similarly affected; old iritis, hypermetropia, internal strabismus, slight nystagmus, interstitial keratitis, especially of right eye, which prevents illumination of fundus; disseminated choroiditis of left eye, one large yellow spot surrounded by pigment near "yellow spot," disc hazy. No nodes anywhere. No enlargement of liver or spleen. Discharged in two months, not much improved.

From a consideration of the preceding cases, we are led to the conclusion, that hereditary syphilis is a more frequent factor in the production of mental disturbance than has hitherto been recognised. As a partial explanation of the supposed rarity of the relationship, the following statements may be noticed:—

1. That congenital deficiency of mind, from inherited syphilis, is probably rarer than mental failure coming on in childhood.

2. That the time at which this mental failure usually shows itself (namely, near the second dentition period) is a time when the manifestations of hereditary syphilis are more or less latent; and if the child has not typical teeth, or some other equally manifest sign, we are not likely, unless the subject is in mind, to think of syphilis. It is, therefore, very important,

in all obscure cases of juvenile dementia, to look carefully for all possible indications of specific taint. Thus, (1) to note the physiognomy and general growth of the child; (2) to examine the long bones for evidence of periostitis; and (3), perhaps most important of all, to examine the fundus of the eye for *choroiditis disseminata.* It may be the only specific sign present; certainly in Case III. it was the first thing that suggested syphilis to me, for the teeth, though changed, were not characteristic. Retinitis and optic neuritis should also be looked for.

Much light might be thrown on this question by thus carefully examining all cases of "eclampsic idiocy," of "epileptic idiocy," of "paralytic idiocy," of "inflammatory idiocy," of "hydrocephalic" and "microcephalic idiocy," especially as I presume it is difficult to get good family histories of cases sent to asylums.

3. It is possible, too, that such cases as those related do not often reach idiot asylums, for the dementia is of an inoffensive type and yet is not likely to be benefited by treatment.

Regarded from a psychological point of view, the preceding six cases, and also those recorded by Drs. Mendel and Jackson, are examples of intellectual rather than of emotional or of volitional insanity; and as in each one of them there is more or less satisfactory evidence that intelligence developed normally up to a certain age, from 5 to 9 years, and then became gradually weakened and in three or four of them utterly destroyed, they probably fall more correctly under the heading of Dementia than under that of Idiocy. It is difficult, no doubt, to draw a sharp line of distinction between the two, but I think it is a question whether juvenile dementia and idiocy have been sufficiently separated by writers on insanity. For example, cases of epileptic dementia are all classed under epileptic idiocy; indeed, I have not been able to find a single reference to a paper on "juvenile dementia."

It would be rash, with the limited number of facts before us, to draw general pathological conclusions with regard to hereditary syphilitic dementia; but the three post-mortems, together with the known pathology of nervous affections from congenital syphilis, enable us to formulate the following propositions:—

1. That the growth of the brain may be hindered by a thickening of the cranial bones, as a result of syphilitic osteitis in early life; Case I. illustrates this in the most striking manner.

2. That it may be hindered by thickened membranes; a chronic meningitis being often started, we believe, by syphilitic periostitis.

3. That thickening and narrowing of the brain arteries, beginning usually as an endoarteritis, are probably by far the most important causes of atrophy of the brain. As Heubner says:[1] "The blood, therefore, moves more slowly and with less tension in the network of the pia mater and in the brain capillaries. Oxygenation is diminished, and the nervous elements suffer a loss in their functional capacity. This effect, if the cause continues, can be no temporary one, and must show itself in symptoms of longer duration."

We know, too, from the observations of Dr. Thomas Barlow,[2] Chiari,[3] Heubner, and others, that syphilitic arteritis is met with in young infants, and may lead to complete occlusion in a child a few months old.

4. That atrophy of the large nerve-cells of the convolutions is sometimes the result of sclerosis of the cortex, set up by some of the morbid processes above mentioned; this is well illustrated by Case II., and also by Dr. Gee's case.

5. Case IV. suggests that occasionally deprivation of one of the principal senses, as hearing, may, by hindering the receptivity of the brain, predispose to dementia.

With regard to treatment, Dr. Hughlings-Jackson remarks in the paper previously quoted, "It would be as reasonable to expect to rid the pupil of lymph effused in an acute attack of iritis, months before we saw the patient, as to restore a brain damaged by syphilitic changes which were active months before. For general cerebral atrophy, consecutive on local syphilitic disease, we could do nothing."

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[1] 'Ziemssen's Cyclopædia,' vol. xii. p. 332. London, 1877.

[2] 'Path. Trans.' London, 1877.

[3] 'Wien. med. Wochenschr.' 17, 18. 1881.

The only hope we can cherish is, that by accurate observations on brain affections from hereditary syphilis we may be able to recognise the disease in an early stage, when only can treatment be of much avail.

OBSERVATIONS ON A CASE OF CEREBRAL CORTICO-MEDULLARY GLIOMA.

BY DAVID FERRIER, M.D., F.R.S.,

Physician to King's College Hospital, and National Hospital for the Paralysed and Epileptic.

ONE of the most significant indications of cortical disease is the occurrence of unilateral spasms—Jacksonian Epilepsy—limited to the face or a limb; or if not altogether limited, commencing always, or nearly always, in the same region, and invading other muscular groups in a certain definite order. If the spasms begin in the face, they next spread to the arm and then to the leg; if they begin in the hand, they attack the arm and then the face; if they begin in the leg, they attack the arm next and the face last. Such is the rule as established by the observations of Hughlings-Jackson.

The diagnosis of cortical disease is rendered certain if, following these more or less limited convulsive symptoms, paralysis of motion without affection of sensibility should occur in the regions formerly convulsed; and still more sure if the paralysis, instead of remaining a monoplegia, should gradually extend and become a hemiplegia. The occurrence of aphasia, when the disease is in the left hemisphere, is another distinguishing feature of the cortical—including subcortical fibres—seat of the lesion. I have recorded many examples of these facts in my "Lectures on the Localisation of Cerebral Disease," and more recent literature affords many other illustrations of the same. The following case illustrates and confirms the principles of localisation and diagnosis of cerebral lesions which I have previously laid down, and at the same time presents other features of considerable interest. It illustrates well the affections of speech in cerebral disease, which have been so philosophically handled by Hughlings-Jackson in the pages of this Journal.

The patient, J. W. L., whose case is the subject of these remarks, first came to my out-patient clinic at the National Hospital for the Paralysed and Epileptic on November 10th, 1882, and was admitted on the same day as an in-patient under the care of Dr. Hughlings-Jackson and myself. He was a man of forty-four years of age, with a quick excitable manner, and of considerable intellectual power. He gained a living by teaching mental arithmetic, and in this he exhibited extraordinary proficiency. During the preliminary examination in the out-patient room he professed to be able to multiply or divide any sum or sums, however large or full of fractions, by a very simple mental process, and also to be able to teach others to do the same. He certainly gave ample proof of his own powers, for, on testing him, he would give the result of a complex multiplication, after looking fixedly at the ceiling for a few seconds, and always quite correctly.

Previous history.—Has lived a rather fast life in the way of alcoholic and sexual excess. Had a chancre, followed by a skin eruption, twenty years ago. For this he was salivated. He married afterwards, and has nine healthy children. His wife had no miscarriages. Seven years ago he used to have attacks of giddiness and confusion. On one occasion he fell down and struck the left posterior parietal region. He does not know whether he was altogether unconscious during this attack. Ten weeks ago he had an attack, and fell down, owing, as he said, to his "brain being overworked." Since then he has felt weak in the right leg, and to some extent in the right arm.

The right arm and leg have been subject to spasms, coming on as often as twenty-four times in the course of the day, apparently without loss of consciousness; but he has also occasionally during the last six weeks had attacks in which he thinks he has lost consciousness and frothed at the mouth.

Condition on admission.—The patient walks with marked lameness of the right leg. He can just get along without a stick. All the movements of the right leg are very feeble, and he cannot move the toes, or invert, evert, or perform plantar or dorsal flexion of the foot. The left leg is unaffected. The tactile sensibility of the right leg and foot is altogether unimpaired, and he feels and localises a touch as well on the right as left.

The right arm is not perceptibly affected, but he says that frequently he has had momentary loss of power in the right arm, so that he would drop his cue when playing billiards.

The resident medical officer—Mr. H. Montague Murray—observed several spasmodic attacks on the right side. They appeared to consist in gyratory or circumductive movements of a deliberate character of both limbs, at the shoulder and hip-joint. The face was not affected, and there was no loss of consciousness.

On other occasions the spasms were seen to begin in the leg and sometimes in the arm, sometimes in both at the same time. When the leg was affected, the spasms began always in the thigh, never in the foot; and when the arm was affected, always in the upper arm or forearm, never in the fingers.

The tactile sensibility of the soles of the feet was equal, but the cutaneous plantar reflex of the right foot was either not obtainable, or much less marked than the left. There was no rigidity of the leg, but the right patellar-tendon reaction was much greater than that of the left. The two sides of the face were quite symmetrical in their aspect and movements.

The patient's general health was fairly good, and there was no discernible abnormality in regard to his organic functions. The patient did not complain of headache or sickness. But percussion or deep pressure exerted on the left parietal region close to the sagittal suture, in the neighbourhood of a perpendicular line drawn upwards from the external auditory meatus, caused pain and wincing, not induced by similar percussion or pressure elsewhere on the cranium.

The general and special senses were normal, and there were no indications of optic neuritis, present or past, on the most careful ophthalmoscopic examinations.

Progress of the Case.—A note on November 22nd records that the spasms affecting the right thigh or upper arm, or both, have continued at the rate of from twenty to thirty a-day.

There has been no loss of consciousness during the fits, and the hand, foot and face have not been affected by the spasms. It was observed to-day that speech was affected to some extent. The patient would in talking, suddenly stop short, and be unable to continue his sentence. In trying to repeat the Lord's Prayer, he could not get beyond "give us"—and then grasped convulsively at his throat, as if there were some obstruction he would remove.

The right arm is now distinctly weak, and he can raise his hand to his head only slowly and gently. But the grasp of the hand is not particularly affected, as he squeezes the dynamometer to 50 lbs. with either hand. There is now also some weakness of the right side of the face, the right naso-labial furrow being less marked than the left when he is asked to show his teeth. The tongue seems to deviate somewhat to the right. The right leg is much more paralysed than before, the foot being absolutely paralysed in all its movements. At this time a careful testing of the tactile sensibility of the two feet showed that, as regards the power of perception and localisation of the slightest touch, the right foot was in every respect as acute as the left. Also as regards the so-called muscular sense of the limb, it was found that the patient could tell with the utmost precision whatever position his toes or foot were placed in. Thus he would say correctly whether his toe was bent or extended; or whether his foot was flexed or pointed, and whether his leg was bent or extended, and the like. There was absolutely no impairment in this respect in the right as compared with the left side.

There was no cutaneous plantar reflex in the right foot, though well marked in the left. The right knee-jerk was greatly increased, and ankle-clonus was now readily obtainable in the right.

The fits continued to have the same character as before, beginning mostly in the right thigh.

On December 21st the notes record that the patient's speech has become more and more liable to the interruptions formerly mentioned, the aphasic attacks lasting longer also than before, and never entirely disappearing. The right leg is now dragged helplessly when he tries to walk, which he can only do when supported. Cannot now raise his right hand to his head, and the grip of the hand is much impaired, so that he can only squeeze 5 lbs., as compared with 50 previously. At this time his speech was affected in a most noteworthy manner. He had the utmost difficulty in finding words to express himself, or even in repeating what was said to him, but as soon as he was given a problem in mental arithmetic he calculated with the utmost facility, and stated the answer without hesitation in appropriate words. Yet if asked to repeat what he had just said, he could only look blankly, and seemed unable to utter a word.

On January 3rd there was total paralysis of the right arm, and paresis of the right side of the face. The right leg was still more paralysed than before. At this date the tactile sensibility was again carefully tested, with the result of showing that the patient was perfectly aware of a touch with the point of the finger everywhere on the right side, as indicated by his gestures, but, owing to his aphasia, no definite answer could be got as to the exact localisation. A slight pinch of the fingers or toes made him wince and cry "Oh!" The cutaneous and tendon reflexes were as before mentioned. An examination of the eyes to-day by Dr. Hughlings-Jackson showed the optic discs perfectly normal, and free from all indications of neuritis.

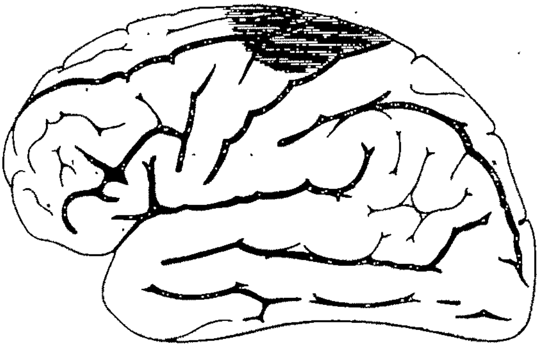
On January 8th the patient's aphasia was much more complete, and his power of calculation also deficient. For a few days previously, when asked to calculate he always returned the answer "fifteen" to every problem. On the 9th he seemed to be able to calculate better, but not correctly, though he had no recurrent utterance "fifteen" as before. The patient's mental condition generally was somewhat obtuse, and there was a tendency to drowsiness. On January 17th this mental obfuscation was more marked, and the patient did not seem to realise his situation, as he occasionally got up, and proceeded to micturate against the wall of the ward.

He is now almost completely aphasic. He cannot name things, or even repeat correctly what is said to him, and in trying to express his wishes stammers and stops. Yet when he was asked, "How much are 4 times 7?" he at once without hesitation said "Twenty-eight." But though he gave an answer promptly to more complicated sums, he almost invariably blundered. He seemed deficient in comprehension to everything said to him, except as regards sums. An examination of the sensibility and reflexes at this date showed unimpaired tactile sensibility of the right side, with abolition of the plantar, cremasteric and epigastric reflexes, on this side. The patient died on January 22nd. No marked change had occurred in the symptoms. He gradually become more obtuse, but he was able to multiply small sums, and give the answer correctly, after he was unable to say anything else, even his own name. He became unconscious six hours before death.

Autopsy, sixteen hours after Death.—Rigor mortis was well marked in the lower extremities, less so in the upper extremities and face. It was equal on both sides. The scalp and skull were normal in appearance. On removal of the skull-cap, it was found that the left side was thicker than the right, in the parietal region, to the extent of an eighth of an inch. The dura mater had a normal appearance externally on the two sides. It was found to adhere more firmly on the left than right, and especially over the region of the upper extremity of the ascending frontal convolution and base of the superior frontal, where it could not be separated from the pia mater without tearing and laceration. The pia mater did not strip readily anywhere from the left hemisphere, and at the base of the superior frontal convolution and neighbourhood could not be separated at all without laceration of the subjacent cortex. Over this area the cortex had a peculiar finely papilliform aspect, like a very close-set cauliflower, and the whole area was firmer to the touch than the rest of the cortex. The abnormal condition occupied the base of the superior frontal convolution and corresponding portion of the ascending frontal up to, but not extending across, the fissure of Rolando. It extended over the margin of the hemisphere for three-eighths of an inch on the internal aspect. Both the ridges and sulci of the region indicated were invaded by the morbid growth. The position of the lesion is indicated in the accompanying figure.

The rest of the brain was normal.

There was nothing specially requiring mention in the abdominal or thoracic viscera—all being fairly healthy.



On frontal sections being made of the left hemisphere, it was found that the cortical region invaded formed the base of a cone-shaped gliomatous infiltration—scarcely to be called a tumour, as it chiefly occupied the place of the healthy tissue—which extended from the cortex through the medullary fibres of the corona radiata to the point where the corpus callosum radiates into the hemisphere.

It did not extend into the internal capsule, for this and the basal ganglia were perfectly normal in appearance. The infiltration had a reddish livid aspect. On microscopical examination of the invaded region—which was carried out by Mr. Murray—it was found that the whole area invaded, viz. the cortex and subjacent medulla, had a similar appearance. It consisted of a finely fibrillated stroma, dense and without interspaces, staining faintly with logwood and carmine; with a parenchyma of cells, of a roundish, oval or fusiform shape. The size of the cells varied. The round cells were smallest, measuring from $\frac{1}{2000}$ in. to $\frac{1}{3500}$ in. in diameter. Each cell had a single small nucleus, staining deeply with logwood. The cells were uniformly distributed, with a tendency to form groups of half-a-dozen or more. At places the cells were aggregated into colonies, each colony consisting of two or three scores of cells, chiefly of the round and oval varieties. The area occupied by the stroma in the centre of the mass was at least double that occupied by the cells. The proper structure of the cortex and medullary fibres was obliterated. Here and there a few pyramidal cells only could be distinguished, but they were small and misshapen.

The tissue was very vascular, and the walls of the capillaries and arterioles seemed thicker than normal. Nothing was found in the cord, except small globules, probably myelin, scattered throughout.

The diagnosis which I made in this case, viz., the existence of a cortical lesion at the upper extremity of the fissure of Rolando, growing downwards, was verified by the post-mortem examination; but the hypothesis I formed as to the nature of the lesion, viz. that it was of a syphilitic character, proved incorrect. And yet with such a definite history of specific disease, and such an assemblage of symptoms, no hypothesis as to the character of the lesion could seem to have better foundation. This is an error which may be of value if it serve to inspire caution against a tendency, too prevalent at the present time, to conclude that diseases of the nervous system are always of a specific nature, because there has been syphilitic infection. I do not know any features which would serve clearly to distinguish between a purely cortical or superficial lesion and one penetrating, as in this case, deeply into the medullary fibres. And yet it would be of the utmost importance if such a criterion could be found, in view of the possible, and perhaps not far distant, treatment of cortical lesions by operative procedure. But to render this treatment at all justifiable, we must have arrived at perfect precision in our localisation. An exact localisation in the case of a morbid growth, such as a tumour, is obviously very precarious, as the area of irritation and disturbance of function is not by any means coincident with the seat of the tumour. When, however, the irritation gives place to very definite monoplegia, we can arrive at the seat of the lesion with considerable accuracy. It was a notable feature in this case that there never were, during the whole course, any of the characteristic symptoms of cerebral tumour, neither headache, nor sickness, nor optic neuritis. I attribute this to the fact that the morbid growth was not in reality a tumour, and did not encroach on the intercranial space, for it merely took the place of the normal tissue, if one could judge by the entire absence of any appearance of dislocation or distortion of the parts. Yet there was in this case, as in a very large proportion of cortical lesions, very distinct pain on percussion or deep pressure over the seat of the lesion. Here, however, there was a complication. It was noted that the skull was somewhat thicker in the neighbourhood of the lesion, and this perhaps may have resulted from the fall which the patient had seven years before. Whether there was any relation between the blow on the head and the development of the morbid growth is a matter of speculation; but the facts seem to me rather to favour the idea that the fall was the consequence of an epileptic attack, due probably to the already commencing disease. Though the convulsive attacks latterly were all of the monospastic or unilateral character, without loss of consciousness, yet it is not unusual to have epileptic attacks of the usual type during the course of disease of the cortex. A patient I recently saw in consultation, who has right brachio-facial paralysis, the termination of a long series of spasms limited to the hand and face, and without loss of consciousness, had a regular epileptic attack, with sudden loss of consciousness at the outset of his disease. He has had one or more since, quite different from the ordinary brachio-facial spasmodic attacks. These have been succeeded by paralysis of the right arm, with perfect retention of tactile and muscular sensibility, and paresis of the face, and less so of the leg.

It is of importance to note in this case the march of the spasms which occurred so frequently. It has been observed by Hughlings-Jackson that, in unilateral spasms, those movements are first discharged which are of the most volitional and independent order. Thus, spasms beginning in the upper extremity commence as a rule in the thumb and index-finger. But I have called attention[1] to the fact that this is not always the case, and that the centres for the various movements of the extremities may be discharged in a different order, varying with the centre in which the irritation concentrates itself. Dr. Jackson has himself recorded a case[2] in which the lesion was situated almost exactly in the same region as the present, in which the spasms began in the shoulder, and thence spread downwards. This was notably so always here. The hand was rarely if ever affected, the spasms being limited to the shoulder movements when they affected the arm. But the thigh was more frequently the seat of the spasm.

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[1] 'Localisation of Cerebral Disease.'

[2] 'Med. Times and Gazette,' June 5, 1875.

A comparison of the region here affected with the position of the motor centres in the brain of the monkey, shows that the centres specially involved are those in which I have localised movements of the leg and arm (centres 2, 3, 4, 5),[1] and the paralysis which succeeded the spasms affected first the leg, then the arm, and the face only partially. The extent of the paralysis, however, in this case was greater relatively than would be accounted for by mere destruction of the cortex to the extent apparent superficially. This was due to the medullary extension of the glioma—the exact extent to which the respective fibres of the various centres of the cortex were implicated being impossible accurately to determine. But the order in which the paralysis occurred, viz. the leg first, then the arm, shows that the disease commenced first in the leg-centre close to the longitudinal fissure, and, by extension downwards, affected the centres for the arm next.

The character of the paralysis is noteworthy. It was confined absolutely to motion, tactile sensibility and the muscular sense being unimpaired. This is the true character of cortical paralysis. Though some writers, more from theory than exact clinical observation, assert that sensibility is affected in lesions of the motor zone, this has no satisfactory foundation. Clinical records have been ransacked for instances of cortical lesion in which tactile and muscular sensibility have been impaired, but with little success. At most, only a few exceptional cases of an evidently complicated nature have been found, many of the observations and arguments advanced being of the most feeble character. Thus Petrina[2] records among the cases of this kind one in which there was a tubercle the size of a hemp-seed (!) in Broca's convolution, surrounded by minute extravasations. This lesion is credited with having produced, in addition to aphasia and tonic spasm of the right arm and right angle of the mouth, anæsthesia of the right side of the trunk, and hyperæsthesia of the right arm. Criticism of such and similar arguments seems unnecessary when the same author records other instances of extensive destruction of the motor zone in which sensibility was absolutely unimpaired. It may be stated that in pure uncomplicated lesions of the cortical motor region tactile and muscular sensibility are unimpaired. If they are affected, the cause is to be found in some complication of the sensory tracts or centres.

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[1] 'Functions of the Brain,' fig. 28, p. 142.

[2] 'Neurologisches Centralblatt,' 1882. (Abstract.)

With absolutely unimpaired tactile sensibility, however, as in this case, and in many which I have investigated and made the subject of clinical demonstration, there is diminution and frequently abolition of the cutaneous reflexes. This fact has been erroneously interpreted by Schiff and some other physiologists as an indication of loss of sensibility, with which, however, it has nothing to do. The personal testimony of a patient on a question of this kind is better than any inference from the lower animals and their behaviour.

The condition of the patient's speech was one of the most interesting features in this case. With the progress of the disease the power of verbal expression, which at first merely suffered occasional interruptions, became more and more impaired, until in the end the aphasia became so great that the patient could not utter his own name. And yet up to the last it was only necessary to suggest an arithmetical problem to him to set the process of calculation in action, and cause the result to be expressed without hesitation in the appropriate words. This shows how organised and automatic the mental arithmetical faculty had become in the man, so that though volitionally he was powerless to express himself in words, he could do so like a machine when set a-going by a few numbers in his ear. Only for a short time did he fall into the one recurring utterance to all problems, viz. "fifteen," and just before his death, when he was otherwise completely aphasic, he could multiply small numbers correctly. But for some time previously he had generally made mistakes in more complicated sums; no doubt the expression of his defective internal speech, which would be more necessary than in the simple sums of the multiplication-table.

INCO-ORDINATION.

BY CHARLES MERCIER, M.B.

"THERE is no paralysis, but there is inco-ordination," is a common phrase. What does it mean? It is considered a complete description, and in great measure an adequate explanation of a motor defect, to say that it is "a loss of co-ordination." But what is co-ordination? To say that a patient cannot walk because he has lost the power of co-ordinating the movements of his legs, is like saying that opium causes sleep by means of its soporific virtue. Unless we can attach some meaning to the term inco-ordination much more definite than is ordinarily attached thereto, we are guilty of what Comte called a metaphysical explanation, or what may perhaps be better termed a verbal explanation,—an explanation which solves the unknown in terms of the still more unknown, and so is no explanation at all. Until quite recently, the word inco-ordination has occupied in medical terminology the position formerly occupied for so long by the term "reflex action." It has been applied to the residuum of unexplained things, and has served in lieu of an explanation. Of late, however, it has begun to be hewn into shape, and the true meaning of a very important term is beginning to emerge from the matrix in which it potentially though not actually existed, as a statue exists in a block of marble. It is no longer regarded as an isolated function, like that of coughing or sneezing, resident in, and confined to, a particular region in the cord or cerebellum. It is no longer supposed that, when a centre in the brain has issued the discharge necessary for a certain movement, this current of force has to go through some subordinate centre to be "co-ordinated" before it is passed into the muscles. It is pretty generally understood that as co-ordination exists in every movement, so it forms part of the function of every motor centre. But still it does not appear to be quite determined what precise element in a movement co-ordination is, or what precise portion it forms of the functions of a nervous centre; and an attempt to determine its nature still more accurately may therefore be of service.

Muscles act, not singly, but in groups. Every movement of a limb, for instance, is brought about by the contraction, not of one muscle but of several, it may be of many; and involves the relaxation, or rather the elongation, of several more. The actual movement of the limb is therefore the resultant of the movements of the muscles concerned. Suppose that the contractions of three muscles pulling in different directions, and each acting with a certain force, produce movement of a limb in a certain direction. If now the action of all the muscles is enfeebled, and all are enfeebled to a proportionate (not an equal) degree, then the resulting movement of the limb will be in precisely the same direction as before, but it will be less forcible. But if one of the muscles is paralysed, the pull in one direction being absent, the movement of the limb resulting from the action of the remaining two will be in a new direction, and will differ in force from the former movement. And if this muscle, instead of being paralysed, is merely weakened, the others remaining as before, the movement of the limb will still be altered in direction, though to a less degree. Or, on the other hand, if one muscle of a set contract too forcibly, the resultant movement of the limb will be in a new direction. So that when a movement is enfeebled, all the muscular contractions that go to make up that movement must be proportionately (not equally) enfeebled; and when a movement takes a wrong direction, it is because the muscular contractions that produce that movement are combined in wrong proportions—are wrongly adjusted—are inco-ordinated. In this case, therefore, an inco-ordinated movement means a movement whose component contractions are combined in wrong proportions.

Let us now take another case of inco-ordination. The movement is in the right direction, and is of appropriate force, but it is carried too far. Such a movement is seen in stringhalt. What is the defect in such a case? By hypothesis the movement is in the right direction, and therefore the several muscles that produce it must contract in proper proportions, —must be co-ordinated in so far as the force of their contractions is concerned. But they all contract too far, the relations among them remaining undisturbed. Now, other things being equal, the extent of a contraction depends on the time that it continues; and the duration of a contraction remaining constant, its extent depends upon its rapidity. Hence, if a movement is carried too far, it is either too long continued, considering its rapidity, or it is too rapid, considering its duration. In other words, the rapidity and the duration of the movement are out of proportion to one another.

Take another very simple form of inco-ordination,—Tremor. Tremor is normal muscular contraction magnified in Time. We know that if a strand of india-rubber is nicked at intervals with a knife, so long as there is no tension upon it, the cuts are imperceptible; but directly the cord is stretched, the cuts gape; and the longer it is pulled, the wider do the notches become. Much in the same way, the several simple contractions that compose a muscular action are imperceptible so long as they remain in close apposition to one another, but when they occur more slowly—when the time over which they are spread is stretched—then the gap between one contraction and the next becomes apparent. In other words, tremor occurs when the number of simple contractions is too few for the time over which they are spread—when the number in a series is out of proportion to its duration.

The movements in tabes dorsalis are wrong in direction, excessive in extent, and wrongly proportioned in time. In each respect they come under one of the previous headings, and the sum total of the defect may therefore be expressed as a combination in wrong proportions of the forces, times of occurrence or durations of the component muscular contractions. The movements in chorea, widely as they differ from those in tabes, agree in being inco-ordinated movements, agree in being wrong in direction, excessive in extent, and wrongly proportioned in time, and agree therefore in being expressible as combinations of muscular contractions in erroneous proportions. In stammering, some sounds, that is some muscular actions, are too prolonged; others do not come into play soon enough; and others occur at wrong times; and in each case the defect is that the times of the actions are combined in wrong proportions.

Now take a much more elaborate case of inco-ordination, such a case as ocular vertigo. In this case, owing to the paralysis of an ocular muscle, the position of external objects as ascertained by sight is misinterpreted; the floor, for instance, is seen with a different inclination from that which it actually has. Now the compound motor arrangements which keep the body upright serve to give us in another way a knowledge of the position of external objects, and especially of the inclination of the supporting surface. Normally the impressions from the outer world received by these two routes harmonise with one another, and blend into a single percept; just as the two separate images received by the two eyes blend into a single percept; but when, owing to an erroneous interpretation, the group of impressions arriving by one route contradict the group of impressions arriving simultaneously by another route, there results a state of confusion which we term vertigo. Vertigo, then, results from the combination of a normal arrangement with a defective arrangement, in other words, it is due to the combination of motor arrangements in erroneous proportions.

In short, every motor derangement which is not either a simple defect or a simple excess must be an alteration in the proportion of the constituents of the movement to one another. There is no other alternative. If a movement is simply weakened, then all its components are proportionally diminished. If it is simply excessive, then all its components are proportionally increased. If it is erroneous in any other way whatever, it is a case of inco-ordination, and it results from a change in the proportion of the elements or factors of the movement to one another. Be they factors of space, or time, or force, the alteration must be in the relations among them, since no other alteration is possible.

Inco-ordination, then, is alteration of proportion; but not every alteration of proportion is an inco-ordination. We have seen that every variation in the movement of a part, whether a variation of direction, or extent, or rapidity, or duration, is due to a variation in the proportions of the elements of the movement. But the variations in the normal movements of a part are literally infinite. The hand, for instance, can be moved to any one of an infinite number of positions within its range. Yet every one of these movements may be normally performed and thoroughly co-ordinated; so that a distinction must be drawn. Although it expresses the truth to say that inco-ordination is an alteration of ratio, it does not express the whole truth. A limitation is required.

When we compare the altered movements of disease as a whole with the movements of health, we find that there is one quality which is never absent in the latter, but which is conspicuously and invariably deficient in the former, and which indicates broadly the division between them. This is the quality of definiteness or exactitude. A normal movement begins at a definite time, traverses a definite path, and ends at a certain place and at a certain time. Abnormal movements have no such precise limitations. The movement of a tremulous hand begins indefinitely, as an exaggeration of the tremor; its path is vague, wandering and exact. It ends by subsiding into simple tremor. In the normal movement of walking, each leg is moved at a definite time, to a definite extent, to a definite spot, on which it is planted with a certain force; but in reeling or staggering it is impossible to know beforehand, with any approximation to exactness, when the next step will begin, what will be its direction, how long it will last, or how forcible it will be; and the total movement compounded of these several movements is similarly indeterminate; the body sways conspicuously from side to side; the course deviates now to the left and now to the right; and the variations of every kind are irregular, ununiform, unpredictable, indeterminate. Similarly with the movements of handicraft. In a normal movement of prehension, the hand is put forth straight to the object, which is then grasped by a direct and well-defined movement. But when a choreic patient attempts to grasp an object, the hand wanders about in a vague and indefinite manner on its way to the object, and when this is reached, the movements of grasping it are numerous, indirect and indefinite. When a healthy person is told to put his finger on his nose, the finger comes straight down upon a definite spot. But when an ataxic patient attempts the same movement the hand wavers about, and the finger finally comes down, not on this spot or that, but anywhere in the neighbourhood. In normal articulation each word and each syllable has its definite duration; but in stammering there is no exactitude. Long syllables become short, and short ones long, and the lengths of all are uncertain; they become fused together; the outlines are blurred. In still more complex cases of inco-ordination the character of the defect remains the same. Wherein do the purposive movements of a worker differ from the incoherent flounderings of a hysterical girl more conspicuously and more fundamentally than in their superior exactitude? And in what consists the still wider divergence of the shouting and whirlings of acute mania save in their still greater indefiniteness of limitation both in time and space?

Inco-ordination we have previously found to be combination in erroneous proportion. We now see in what the error consists. It is in the substitution of an indefinite for a definite proportion; and the needed limitation is added to our conception of inco-ordination when we define it as *combination in indeterminate ratio;* co-ordination being combination in determinate ratio. Such a definition appears to include everything that co-ordination is, and to exclude everything that it is not. It applies not only to the co-ordination of the elements of movements, to the combination of the spaces, times and forces of the muscular contractions that go to make up a movement; not only to the combinations of movements with one another; but to that combination of impressions with movements which is the highest form of co-ordination, and which constitutes the raw material of Conduct.

Clinical Cases.

CASE OF CERVICAL MENINGO-MYELITIS: EPILEPTIFORM SEIZURES: ATROPHY OF NERVE-ROOTS AND MUSCLES: UNUSUAL FORM OF ASCENDING DEGENERATION IN THE SPINAL CORD.

BY C. F. COXWELL, M.B. (CANTAB.),

Late Resident Medical Officer, National Hospital for the Paralysed and Epileptic.

CHARLES H——,* aged 32, was admitted on October 22nd, 1881, into the National Hospital, under the care of Dr. Ramskill, who has kindly permitted me to publish the case. He had led a very gay life, and had several chancres as well as epididymitis, but a history of the usual symptoms of syphilis could not be obtained. In May 1881, he had "neuralgic" pains in the left eye, and then "rheumatic" pains in the shoulders. In June he noticed that he saw double, the diplopia gradually improving and disappearing before admission. Soon the fourth and fifth fingers of the left hand became weak, so that he could not extend them. In August he gradually lost power in the left leg, and before long could not walk at all, owing to nearly complete paralysis of the lower extremities. In a few days sensibility had departed from the lower limbs, and from the trunk below the waist, at which level there was a sense of constriction. A little later the right arm became powerless. There was involuntary evacuation of both urine and fæces during three weeks, but this symptom had improved when he was first seen by Dr. Ramskill shortly before admission.

When admitted, he was a good deal emaciated. The lower limbs were much wasted and the muscles soft and flabby. He could just manage to draw up the legs a little, the right better than the left; could flex the knees and ankles, but not extend them; could not move his toes. The legs were not rigid, but were kept in a flexed position, to which they returned when straightened. The reflexes were as follows: plantar, both obtained, but with some difficulty; ankle-clonus obtained, right and left, but not very readily; knee-jerk, both excessive; cremasteric, abdominal and epigastric, absent.

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* The early notes of this case were taken by Dr. C. E. Beevor, late Resident Medical Officer.

The upper limbs were like the lower, much emaciated. The left arm was much more wasted than the right; the posterior aspect of the left fore-arm being positively concave: the difference between the right and left hands was equally distinct. While there was free movement at all the joints of the right arm, the power over the left was very much diminished; extension at the elbow and all movement of the fingers being quite absent, while the other actions were very feebly performed. As tested with the dynamometer, the right hand possessed a grasping power of 25 lbs., the left of 0 lb.

As regards sensibility, he could localise tactile impressions correctly on the left foot and leg and on the right leg, but less correctly when touched on the right foot. The sensibility of the rest of the body was good. There was no analgesia. There was no tenderness over, or irregularity in, the course of the spine. He had to obey the call to pass his urine immediately he felt the desire, and his bowels were constipated. The urine was healthy.

He was unable to turn the left eye outwards beyond the middle line, but there was no other abnormality of head or face, and no optic neuritis. There was slight nocturnal elevation of temperature, probably due to an extensive bedsore which exposed a large portion of the sacrum.

Progress of the Case.—The patient suffered greatly from his bedsore, but after a time began to improve. His legs remained drawn up at the knees, at which point they were pressed together with such force as to cause him great pain.

On March 6th, 1882, he could for the first time lift both feet clear of the bed, and his right hand was much stronger. The paralysis of the left external rectus had disappeared some time, and the large bedsore had nearly healed. His left arm was kept habitually in a position of extreme abduction at the shoulder, flexion at the elbow, and dorso-flexion at the wrist.

On May 4th he became suddenly much worse, was delirious, and vomited three times. His temperature rose to 105° and his pulse to 150. There was no optic neuritis at this or at any time. Tremors and spasm of the legs were incessant and caused him to shriek out. The explanation of his altered condition became evident in a few days, when a large bedsore made itself visible through the thick skin of his left heel, and shortly afterwards a large portion of the os calcis became bare. From May 6th until the day of his death, any movement of the right, that is, the non-paralysed arm, caused him excessive pain. On the 12th there was subsultus, and occasional delirium.

On the 13th he was attacked with three fits, which are of interest, on account of the fact that the only lesion discoverable at the autopsy was spinal. The first fit occurred at 7.30 A.M., and lasted a few minutes. It affected at first only the left arm and leg, and the left side of the face, and later the right side of the face and right arm and leg. The head and eyes at first were turned to the left, later to the right. The second fit lasted also a few minutes, and was exactly the counterpart of the previous one, with the difference that it began on the right side and terminated on the left. The above facts I wrote down, as they were given to me by an attendant on whose correctness of observation I can rely. Consciousness was retained during the second fit, but not during the first. When I first saw him, at 8.30 A.M., the third fit had been half an hour in progress. I took the following note: "Patient is lying on his back; head turned to the left; eyes open. There are small clonic spasms of the eyes from right to left; also of the mouth, chiefly on the left side; also of both fore-arms, both of which are nearly completely flexed. The fingers of both hands are semiflexed and rather rigid, but not affected by clonic spasm. The right leg is not at all affected. There are clonic spasms of the left foot from the ordinary position to that of dorso-flexion, and the leg is so thin that the tibialis anticus and extensor longus digitorum can be seen to contract with each spasm. The respirations are 120 per minute, and very noisy: the breathing is entirely abdominal. The pulse is 120 and very weak. The pupils are much dilated and equal. The face is sweating and very flushed. He is not unconscious."

Two hours later I continued my note: "Is still in the same epileptiform convulsion. Is conscious. The muscles affected are the right sterno-mastoid, the face being turned upwards and to the left, the levators of the eyelids, the left external rectus and the right internal, the flexors of the left thigh, the hamstrings on the same side, and the dorso-flexors of the left foot. All these muscles are subject to clonic spasm, occurring at the slow rate of two a second, as often seen at the end of an ordinary epileptic seizure. The left transversalis abdominis is affected, but not the diaphragm. The respirations are now at the rate of forty a minute." This fit lasted until 12.30 P.M., that is, four and a half hours.

The patient continued in the typhoid condition, and in two or three days developed signs of pneumonia. He died on May 19th. The treatment employed by Dr. Ramskill consisted in giving large doses of iodide of potassium, and nerve tonics.

The following are the chief results of the post-mortem examination, which was made 12 hours after death. Rigor mortis, not well developed; body much emaciated. There is extreme atrophy of the muscles of the left hand and arm, and small portions of muscle removed from the left thenar eminence and the extensor surface of the left fore-arm are very pale compared with corresponding specimens from the right arm. The spinal cord is removed without any difficulty, although there are slight adhesions on the left side between the dura and the vertebræ in the cervical region. The dura mater is a good deal thicker than normal in the region of the cervical enlargement. Here over a vertical extent of about three inches the arachnoid and subarachnoid cavities are obliterated by delicate fibrous tissue, chiefly placed laterally around the roots of the nerves, the obliteration being much more complete on the left side than on the right. It is difficult also, both anteriorly and posteriorly, to separate the dura mater from the cord. Both the dura mater and the adhesions are perfectly pale. More exactly the adhesions between the dura mater and the pia extend to the left side from the level of the fifth cervical to that of the second dorsal nerve-roots. The posterior roots on the left side of the nerves between the fifth cervical and second dorsal inclusive are markedly atrophied. The anterior roots of the fifth and sixth cervical nerves are very distinctly atrophied, being hardly more than mere threads. The atrophied nerve-roots are of a dirty-grey colour. On cutting consecutive transverse sections of the cord, the whole of the cervical region, except the highest part, is found to be softer than normal, while over the central and lower two or three inches its substance is nearly fluid, of about the consistence of cream, and of a dirty-white colour. The softening is much more marked in the region of the grey matter than in that of the white, and more on the left than on the right side. The dorsal region of the cord is somewhat atrophied, and the lumbar greatly so, it being hardly larger than the dorsal, and nearly all trace of the lumbar enlargement having vanished. The brain presents no abnormal appearances beyond marked venous congestion, and careful examination fails to detect anything wrong with the cranial nerves. The arteries at the base of the brain are of healthy appearance. The right lung is somewhat œdematous, and its lower lobe congested. The left lung contains pneumonic patches scattered through it. In its lower lobe the consolidation is more general, and a small portion of it cut off and thrown into water sinks. The heart is healthy; the right side is full of black clot; the left side empty, except for a little pale clot. The liver, kidneys, and spleen are normal. In none of the organs are there any gummata or other signs of syphilis.

Even to the naked eye very marked degeneration in the spinal cord was visible after it had been hardened in Müller's fluid. In the upper cervical region, situated between the columns of Goll and the posterior cornua, and quite distinct from them, were on each side nearly symmetrical tracts of sclerosis. That on the left side was of extreme distinctness, and reached from a little behind the posterior commissure to the posterior margin of the cord. The columns of Goll were sclerosed, but far less densely than the above-named tract on the left side. In the dorsal and lumbar region the antero-lateral columns were sclerosed in the crossed pyramidal tracts, the left a good deal more so than the right. Under the microscope the degenerated tracts exhibited the appearances of dense sclerosis. It is to be regretted that owing to a mistake the medulla was not saved. The microscopical examination of the portion of muscle removed from the left arm showed that many of the fibrillæ were much atrophied. They presented a wavy outline. In some of them the striation was preserved, in others it was very indistinct. There was neither fatty degeneration nor increase in the amount of connective tissue. The normal structure was preserved in the specimens of muscle from the right arm.

Among the numerous interesting features of this case are the epileptiform seizures which occurred a week before the patient's death. In connection with them it may be noted that there was no lesion discoverable in the brain to account for them, and that the patient had never before had any fit. Also that they were of a distinctly unilateral type, and consciousness was at least in two out of three of them not lost. A case reported by Dr. Gowers in vol. xxvii. of the 'Transactions of the Pathological Society,' presented in many respects similar features to the present one. It is that of a young man, aged 30, who, for three months before death, was subject to severe convulsive seizures, commencing unilaterally in the left hand. The patient died in one of these attacks. At the post-mortem the brain was found to be healthy, but there was postero-median sclerosis in the whole extent of the cord, most dense in the lumbar region. The patient had exhibited no spinal symptoms. The region between the columns of Goll and the posterior cornua was healthy. Charcot states, at page 116, vol. ii., of his 'Lessons on the Diseases of the Nervous System' (New Sydenham Society's Translation) that he has collected five cases of compression of the cervical region of the cord, and five cases of compression lower down, in which there were epileptic seizures. It is to be presumed that in some of them at least there was ascending degeneration of the usual type. In one of these cases the pressure was exerted rather on the bulb than the cord. Was irritation of the medulla the explanation of the epileptic attacks here alluded to, and also of those in the present case?

With regard to the peculiar position of the patient's arm and hand, which is the same as that described by Professor Charcot as occurring in pachymeningitis cervicalis hypertrophica, when the lesion is situated in the lower part of the cervical enlargement, it may be remarked that the softening was diffuse in the present case, and certainly included the lower part of the cervical enlargement. In addition, the adhesions on the left side extended between the level of the fifth cervical and second dorsal nerve-roots. Dr. Ferrier states, in the number of 'BRAIN' for July 1881, that he found that stimulation in the monkey of the sixth cervical anterior nerve-root caused adduction and retraction of the upper arm, and extension of the fore-arm, with pronation and flexion of the wrist, and that stimulation of the fifth anterior root caused extension of the fingers and wrist, supination of the wrist, drawing of the hand to the mouth, and flexion of the fore-arm. It may be reasonably expected that in the atrophy of the fifth or sixth nerve-root the arm would take a position the opposite to that which it assumes when either single root is stimulated. But in simultaneous atrophy of the two roots, a position in some respects agreeing with that deduced as likely to occur in atrophy of the fifth root, and in other respects with that to be expected in atrophy of the sixth root, would be looked for. The patient's left arm was in such a position. It was abducted at the shoulder, and the hand was semiprone, as might be expected in simultaneous atrophy of the fifth and sixth roots. It was flexed at the elbow, and the wrist was extended, which would be expected in atrophy of the sixth root, but not when the fifth was affected. All the muscles of the left arm were wasted to an extraordinary extent. This agrees with the greater degree of softening on the left side, and the atrophy of the left nerve-roots.

On seeing the spinal cord in the hardened condition, Dr. Bastian at once recognised the sclerosed tract outside the columns of Goll, in the upper cervical region, as exactly similar to a tract of secondary degeneration which he found in a case several years ago, and kindly told me where I should find the case recorded, namely, in the 'Medico-Chirurgical Society's Transactions' for 1867. It is that of a man who died after receiving an injury to the spinal cord, at the level of the fifth and sixth cervical nerves, caused by a fall through a space of twenty-five feet. There was found secondary degeneration of a descending character in the direct and crossed pyramidal tracts, while above the lesion, both in the columns of Goll and symmetrically on the two sides, in a narrow region between those columns and the posterior cornua, there was a separate tract of sclerosis.

It might naturally have been expected that, with the atrophic condition of the posterior roots which was found in the cervical region, the patient would have suffered from partial anæsthesia, at least in the left arm, but such was not the case. The tactile sensibility was repeatedly tested, and never found to be impaired over either arm, or in the region of the chest or back. Nor is this very surprising, if the common clinical experience be remembered of the frequency with which the sensibility in paraplegia both is less affected than the motor power, and the greater rapidity with which, when impaired, it returns than does the latter. That there was neither pain nor tenderness in the region of the spine, and that there were no pains in the arms, such as one might have looked for in cervical meningitis, is an instance of the fact that the classical symptoms of meningitis are not always present. As has been above stated, however, there was severe pain whenever the right arm was moved, for a fortnight before death.

TWO CASES OF ACUTE SPINAL CORD DISEASE.

BY STEPHEN MACKENZIE, M.D.

CASE I.—ACUTE ANTERIOR POLIOMYELITIS IN CONNECTION WITH VARIOLA (POST-VARIOLAR PARALYSIS).

G. K., æt. 56, ship's carpenter. An intelligent steady man. Had had two attacks of lumbago. With this exception, he had had no accident or disease until the present illness. On February 7, 1880, he was attacked with the initial symptoms of smallpox—loss of appetite, thirst and vomiting; but three days previously he had felt cold in the back, in and between the shoulders, although he was able to work. The eruption of smallpox appeared first in the groins, on the 9th, on which day he took to his bed, but was not very ill. Three days after taking to bed (i.e. on the 4th day of eruption) he noticed pain in both shoulders. The pain was of an excruciating darting character, worse at night and subsiding somewhat in the morning. It was so bad as almost to make him cry. Any movement of the body, or resting on the shoulders, increased the pain. About 7 days after the onset of the attack of pain in the shoulders, he felt pain starting between the left ear and nucha and extending down to the shoulder and spine. The eruption, which had not previously appeared at the back of the head, speedily did so (the rest of the eruption had by this time disappeared), and was followed by subsidence of the pain. Soon after this he discovered a want of power in the left arm, which continued up to the time of my seeing him. At about the same time that the pains came on in the shoulder, he observed "a dead feeling" in the upper part of the right arm, and this also persisted. The pain altogether lasted about 15 days, gradually wearing off. When he got up, at about the 17th day, he found himself quite unable to raise the left arm from the shoulder, whilst all movements of the right arm were perfect. He was brought to me by Dr. Dundas Grant, who had carefully watched the case throughout, and who had correctly diagnosed its nature. To him I am indebted for many of the particulars of the case.

The patient was seen by me, March 11, 1880, and on some subsequent occasions.

It was observed there was sinking of the supra-spinous fossa and flattening and flaccidity of the infra-spinous fossa; the superior angle of the scapula, when the arm was at rest, approximated too near to the spine, and the spinal border sloped obliquely away from it. The scapula appeared to cling to the thoracic wall; the inferior angle not standing out so prominently as its fellow. The muscles around the acromion were not so plump as on the right side, and felt flabby. There was semi-luxation of the head of the humerus, the head of the bone dropping, and being felt unnaturally distinctly below the acromion. The axillary fold was correspondingly lower than on the right side. The movements of the hand and forearm were intact. He could push out in front and pull back the upper arm, but could not abduct the arm in the least; he could adduct it powerfully, he could hold the arm back to a horizontal plane, by stooping, as far as the other, but he could not get the arm in front of him. When, after drawing back the arm he hit out in front, the arm was carried a little forward and then suddenly dropped. When made to grasp a stick, and by its means the arms were raised in front of him, the right scapula stood boldly out from the thorax the whole length of its spinal border, the skin puckering at the inferior angle. The left scapula, on the other hand, did not stand out at all from the thorax, the superior angle was tilted outwards, and the shoulder markedly raised. He could not rotate the left arm outwards so completely as the right. There was an area, 3½ by 4 inches, just below the point of the *right* shoulder, where sensation was very defective. Examination with faradic current showed:—

Diminution of electro-sensibility over *right* deltoid region, and in a less degree around it.

Loss of electro-contractility in left deltoid, supra- and infra-spinous muscles. Well-marked contractility in right deltoid. No loss of electric excitability in the other superficial muscles. Exaggerated contraction to slowly interrupted galvanic current of left deltoid and supra- and infra-spinous muscles.

In order that the patient might have the full benefit of electrical treatment, he was placed under the care of Dr. de Watteville; and after persevering treatment for many weeks the power in the shoulder muscles slowly returned, and the patient was able to return to work.

Remarks.—This case presents many points of interest. In the first place, it is a very characteristic one of acute poliomyelitis. It set in with pain. There was general disturbance to the health, but in this case it was due to variola. Often such cases at their onset are taken for rheumatism, as in a case to be subsequently related. As is usual in acute poliomyelitis the brunt of the inflammation fell on the anterior cornu, as evidenced by the paralysis and wasting of muscles, followed by the characteristic electrical reaction of degeneration. It will be observed that the paralysis involved a certain group of muscles, associated in action upon the shoulder. The deltoid, supra- and infra-spinous muscles, the teres minor and the rhomboid muscles, and possibly the levator anguli scapulæ, were chiefly involved, producing an inability to raise the shoulder or carry forward the arm. The loss of action of the rhomboid muscles—difficult, owing to their depth, to determine by electricity—was shown by the want of posterior fixation of the scapula; so that when the arm was raised or moved forwards, the scapula, instead of being held back parallel with the spine and standing out boldly from the thorax, glided away from the spine, clinging closely to the thorax. It would seem, from the muscles affected by the paralysis, that it was a physiological rather than an anatomical group that was affected. It is clear that the lesion in the left anterior cornu was at the level of the fifth cervical nerve, where the supra-scapular nerves, and the nerves to the rhomboid muscles and levator anguli scapulæ, are given off. The lesion also involved the point from which the circumflex nerve arises, as shown by the paralysis of the deltoid and teres minor, and the loss of sensation in the opposite side of the body.

This brings me to a second feature of interest in the case, namely, the loss of sensation over a small portion of the right shoulder. This was a small well-defined patch in the deltoid region, corresponding to the cutaneous branches of the circumflex nerve. Thus we have coming in concurrently, loss of motion on one side and loss of sensation on the other side, necessitating a lesion of the anterior cornu for the former, and a lesion of the posterior cornu for the latter. It was thought at first, especially by some of the French writers—Charcot, Vulpian, Joffroy—that any involvement of the posterior cornu in poliomyelitis was due to secondary horizontal extension from the anterior cornu. But, as shown by Dr. Barlow,[1] cases by Drs. Charlewood Turner and Frederick Taylor give anatomical grounds for the belief, that the lesion in the posterior cornua sometimes found may be of a primary nature, concurrent with that in the anterior horns; whilst the case I have narrated, from the contemporaneous affection of sensation and motion, unquestionably makes the lesions in both the anterior and posterior horns common and primary. The pain, which was a conspicuous feature at the onset of the affection and which affected the area subsequently paralysed, I take to be due to affection of the posterior horns, or the radicular fasciculi of the posterior columns. This would tend to the belief, that the mischief began quite as early in the posterior as in the anterior part of the cord.

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[1] 'Brain,' Vol. II. p. 289.

We have still further to note the occurrence of the paralytic symptoms in connection with smallpox. The concurrence of paralysis and smallpox has been noticed by different writers. Paralysis accompanying smallpox appears to have been noticed as early as the 17th and 18th centuries; and, according to Grasset,[1] Horstius and Freind observed that it was not limited to grave cases. Sydenham, Mead, Lieutaud, Van Swieten, and Morton also described it. Trousseau, speaking of the rachialgia characteristic of smallpox, wrote "it is not, as has been supposed, a muscular pain, but is dependent upon an affection of the spinal marrow. Here is the proof. In a great many cases (and last year, within a few days, I could have shown you two examples) the lumbar pain is accompanied by paraplegia. Without your putting any leading questions, the patients themselves mention this paralysis; they complain of painful numbness in, and inability to move, the lower extremities. When you inquire whether the upper extremities are similarly affected, you discover that their motor power is in no way impaired. The paralysis sometimes affects the bladder, as evidenced by retention of urine, or at least by great dysuria. The paralytic symptoms are generally of short duration, but in some cases they continue till the 9th or 10th day; generally they cease spontaneously when the eruption appears. There are, however, cases in which the paralysis persists not only during the whole course of the disease, but likewise constitutes one of the complications of convalescence."[2] Paraplegia appears to be the form that has most frequently been met with, but the paralysis in some cases has been accompanied by formication and pain. Roger and Damaschino have described acute spinal paralysis; Kahler and Pick, ataxy; and Berhier, Liouville and Long, sclérose en plaques. Westphal has recorded[3] some cases of variolous paralysis. In one there were tremors, peculiarity of articulation, dysphagia, ataxy of upper and lower extremities, without paralysis. The condition was like that seen in sclérose en plaques. I also have seen, some years ago, two cases of a condition very similar to that observed in Westphal's case, after variola. But Westphal has also described two cases with necropsies, at which a disseminated myelitis, in the one predominated in the grey substance, and in the other in the white substance of the cord. Joffroy has described a case in which the spinal cord was intact, but the nerves affected. Vulpian has described a case of paralysis of the deltoid, and Bailly, paralysis of the arm or forearm, which Grasset relegates to the category of a local neuritis. In the case I have narrated it will be observed, from the muscles affected and the paralysis of motion, the lesion could only be a central one, in the cervical cord.

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[1] 'Traité Pratique des Maladies du Système Nerveux,' 1881, p. 1058.

[2] 'Lectures on Clinical Medicine,' Eng. Trans., vol. ii. p. 51.

[3] 'Berlin. Klin. Wochensch.,' 1872, Nos. 1 and 47.

One other point to which I wish to draw attention is, the delay in the appearance of the eruption in the neck, in the part in which there was localised pain. The eruption had disappeared from other parts, but on the cessation of the pain the eruption appeared over the previously painful area. We sometimes see eruptions of nervous origin, and distributed in the course of nerves; but here is an example of what appears to be an action of an inhibitory nature, dependent on some spinal affection, and which probably is just the opposite of that which causes or permits eruptions of nervous origin or distribution.

CASE II.—ACUTE CENTRAL MYELITIS (?) WITH ARTHROPATHY.

M. H., aged 46, widow, housekeeper to a firm in the City, of temperate habits and an abstainer for last three or four years, admitted into the London Hospital Nov. 12th, 1879. For three weeks before admission she had been feeling unwell, with loss of appetite, chilliness, and sore throat, attributed to cold. These symptoms were followed by pains in the joints. A physician was called in, who pronounced the case to be one of acute rheumatism, and she was sent to the hospital. Her family history was unimportant. The only previous illness was pleuro-pneumonia, 18 years before. Syphilis and gout were denied, and there were no evidences of either.

On admission, the patient, a woman with dark-brown hair and eyes, had a distressed expression of face, as if in pain. She lay with least ill-ease on her back, complained of great pain in the joints (unfortunately the notes do not specify the exact condition of the joints on admission). No abnormal signs existed in the heart, lungs, or abdomen. P. 104; R. 30; T. 102°; saliva and sweat, acid.

I saw the patient on Nov. 14th, two days after admission, when the condition of the joints was as follows:—The dorsum of each foot and the ankle-joints were swollen, red, and pitted on pressure. The redness was somewhat streaky and patchy, and the superficial veins slightly injected; the reddened part was extremely sensitive to pressure. On the dorsum of the left foot was a vesicle passing into a pustule. The skin of the right metatarso-phalangeal articulation was red, but not painful. The left wrist-joint was red; no effusion into joint. I remarked "the appearance of the feet resembles very much an acute attack of gout." The serum was ordered to be examined for uric acid.

Nov. 17th.—The patient complains of pains in all her limbs, and is continually groaning. Her tongue and lips are dry and brown. The joints are not more swollen; the left knee is very painful. The serum from the skin over this knee was not found to contain any uric acid. She was given a quinine mixture on admission; this was changed to an effervescing mixture on the 16th, and to-day mist. colchici alkalin. was ordered. Her temperature in the evening was 100°.

Nov. 20th.—Much more free from pain this day. Joints not so much swollen. Diarrhœa for last two days; urine and fæces passed in bed. Tongue dry in centre. Is delirious.

Nov. 22nd.—Was delirious during the night. Diarrhœa continued; motions passed under her. Morph. hyd. gr. $\frac{1}{3}$ was given at night to procure sleep, with good effect. Ordered a mixture of bicarbonate of potash and liquid extract of bark.

Nov. 24th.—Diarrhœa continued; motions still passed involuntarily. Bedsore forming on sacrum.

Nov. 26th.—Better; pain not so severe; was able last night to raise herself to pass her motion. There is now cystitis, with mucus, and albumen in urine.

Nov. 30th.—No longer delirious. Passes urine and fæces in bed.

Dec. 4th.—Can use bed-pan; only passed urine involuntarily once in 24 hours. No pain in limbs; can raise them a little.

Dec. 8th.—Pain in back and limbs, preventing her sleeping. No longer incontinence of urine and fæces. Mist. potassii iod. (gr. v.) ter die.

Dec. 15th.—Complained that she could not keep her legs still, owing to constant twitchings. Mixture of iron and tincture of bark.

Dec. 18th.—Twitchings of legs kept her awake.

Dec. 23rd.—No albumen in urine. Temperature normal. At this time the lower extremities were examined with galvanism. There was found in the muscles in the front of the legs loss of faradic contractility, and degeneration-reaction with galvanic current, the muscles acting chiefly to the positive pole. These observations were confirmed by Dr. de Watteville.

Jan. 2nd.—Was prevented sleeping last night, owing to pain in left knee. The skin of the heels covered with thick crusts, beneath which there is some sero-purulent discharge.

Jan. 7th.—Left heel very painful, preventing her sleeping.

Jan. 12th.—Improving in every way. Sits up daily for three hours, but cannot walk. Heel painful.

Jan. 26th.—Cannot yet stand alone, but is improving in every way. Is gaining flesh and strength.

Feb. 2nd.—Still improving, but cannot walk.

Feb. 14th.—Can manage to stand alone, and, with aid of a chair, can go up and down the ward. She walks on the toes, being unable to support her weight on the heels.

Feb. 20th.—There is a small ulcer or sinus on the left heel; the skin around is indurated, and the veins are distended. She was ordered crutches. Cod-liver oil, a teaspoonful three times a day.

Feb. 28th.—Left heel better. Manages to get about a little.

March 5th.—Is still improving. Can walk alone a little, and walked last night to chapel without assistance.

March 6th.—Sent to Convalescent Hospital.

No change in fundi oculorum, which were repeatedly examined throughout the case.

Remarks.—Let me gather up the salient points from the foregoing notes. After a week or two's malaise, the patient, a middle-aged woman, complained of sore throat and articular pains, followed by swelling of the joints and feverishness, having some resemblance to acute rheumatism. A few days later the notes show, as regards the character of the arthritis, that it does not resemble what is usually seen in acute rheumatism, but is more like the arthritis of gout. It affects joints of both upper and lower extremities, and the serum of the blood is found free from uric acid. The temperature was somewhat raised, compatible with a moderately severe attack of acute rheumatism or gout. It is then noticed that incontinence of urine and fæces occurs—conditions most unusual, except in very grave cases of rheumatism or gout. The incontinence of urine is followed by cystitis. The patient becomes delirious, and a slight bedsore forms over the sacrum. There are twitchings of the lower extremities. It is found that the muscles of the legs have undergone wasting, and give the degenerative reaction to the galvanic test. The patient is troubled with severe pains in the knee and heels, and painful ulcers form at the back of each of the latter. The fever, meanwhile, has subsided, the arthritis and incontinence have disappeared, and, though left weak, the patient progressively recovers her strength and the power in the lower extremities. The recovery, I believe, has been complete.

In this case there was weakness and atrophy, especially affecting the muscles of the front of the legs, with degeneration reaction, from which it may be inferred that there was some acute cornual inflammation. But the mischief was clearly not confined to the anterior horns, the sensory disturbances, and vesical and fæcal difficulties, the bedsore and the ulcers on the heels (probably originating in pressure-sores), point to implication of the posterior grey horns or radicular fibres. The exact area of the lesion was not determined by the reflexes, but it is probable, from the arthritis of the upper as well of the lower extremities, that it was of considerable vertical extent. I think, therefore, if the case must be fitted into one of compartments into which the train of morbid processes is arbitrarily (but conveniently) divided to form clinical entities, this would rather come under the category of *acute central myelitis.* The preceding case, however, as do others, shows that, in cases which would be generally admitted to be acute anterior poliomyelitis the posterior grey horns do not escape, though the anterior horns may suffer the most. Nor is it likely that an inflammatory disease of a part with such ill-defined vascular territories would be abruptly confined to so limited an area as one horn. It will probably be found, as recorded cases supply a wider basis for inductions, that various intermediate conditions exist between acute anterior poliomyelitis and acute central myelitis.

The point, however, of greatest clinical interest in the case is the occurrence of an acute arthritis, affecting several joints, in association with an acute affection of the spinal cord—an acute spinal arthropathy. The connection between the two appears distinct. Was it the acute febrile disease caused the spinal affection, or *vice versâ?* The preceding case shows the former may be the case, at least in smallpox and some diseases of its class. On the other hand, I know of no instances in which either rheumatism or gout has been attended with this complication, and we do know that affections of the cord are liable by disturbance of trophic centres to give rise to arthritic and osteal disorders. The present case throws no light on the localisation of such centres. It seems to me more probable that the arthritis was a consequence of the spinal affection, and the case strengthens the belief that some of the irregular forms of arthritic disease may own a similar origin.

CASE OF FRACTURE OF THE SKULL IN WHICH CONJUGATE DEVIATION OF THE EYES, WHICH HAD EXISTED FOR FOUR MONTHS, WAS REMOVED BY TREPHINING.

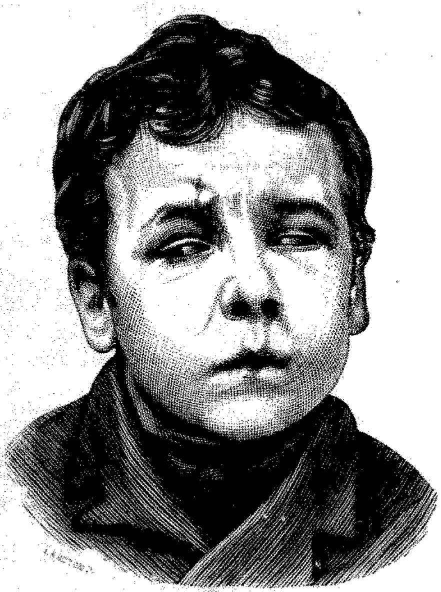
BY GEORGE THOMSON, M.D.

James Lovely, aged 14 years, was admitted to the Oldham Infirmary on January 14th, 1882, under my care. His occupation is that of a "piecer" in a cotton-mill. He was cleaning the "weights" of the "jenny" or "mule," during which operation it was drawn out from the "headstock" and retained in that position by a catch termed the "Samson." The boy displaced the Samson accidentally, and the mule, being released, ran home, catching his head in such a manner that it was struck and compressed between the rounded ends of two bars of iron, between which there would scarcely be room for the doubled fist of a man. The pressure was applied to the head at symmetrical points immediately beneath the parietal protuberance on each side, but the left side of the skull alone was fractured.

On admission, the boy was found to be suffering from the symptoms characteristic of cerebral irritation, viz.—lying curled up on his side with all his flexors contracted, and responding with some irritable exclamation or movement to any attempt to move or rouse him. The eyelids were tightly closed, and resisted any attempt to drag them open; while the pupils were turned out of sight the moment the lids were dragged apart, and consequently could not be seen. No note was made of the direction of the rotation of the eyes. There was a scalp wound, about two inches long, extending vertically upwards over the forehead from the middle of the right eyebrow, and a large contusion, with great subcutaneous effusion but no scalp-wound, above and behind the left ear. There was also bleeding from the nose and mouth.

Soon after admission the temperature rose to 102·5°, the breathing became at times somewhat stertorous, and delirium supervened. The temperature reached no higher point, and finally fell, gradually reaching the normal point on the 21st day after admission. About that time a partial recovery of consciousness took place, the boy passing into a fatuous condition, in which he became so filthy in his habits and expressions that he had to be isolated. This condition continued for about six weeks, during the whole of which time the eyes were never voluntarily opened.

On the disappearance of the subcutaneous effusion a large depressed fracture was observed, one inch below the most prominent part of the parietal protuberance, and two inches above and rather behind the helix of the ear. This depression is about a quarter of an inch deep, and is two inches in the antero-posterior, and one inch in the vertical diameter.



About two months after admission, the boy opened his eyes and became sensible and good-tempered. It was then noticed that both the eyes deviated to the right. He complained of a sense of pressure over the eyes and forehead, but more especially over the left eye. I decided to wait a few weeks to see if time would remedy this condition. Three months after the injury, I observed that the deviation of the eyes was more marked; so much so, as to cause the pupils to disappear behind the canthi, and thus make the boy practically blind. He was sensible of any strong light placed towards his right side, and could tell when any opaque body passed between him and the light, but could not recognise any object. I made an attempt to examine the retina with the ophthalmoscope, which was defeated by the great deviation of the eyes. Four months after the injury, I submitted the case to my friend Dr. James Ross, and on his recommendation I decided to trephine at the seat of fracture in the hope of removing the cause of the deviation of the eyes. The considerations on which Dr. Ross based his opinion will be found appended in his own words.

On June 9th, about five months after the date of injury, I trephined at the apex of the depression, where it was deepest. I found two pieces of the inner table impinging deeply on the surface of the brain, one of which, about half an inch long, came away attached to the portion of bone removed by the trephine, and the other, nearly one inch long by one-third of an inch broad, was afterwards removed at the lower extremity of the opening made by the trephine.

The boy made an excellent recovery from the operation, the wound being completely cicatrised in three weeks. As soon as he recovered from the chloroform he expressed himself as feeling better, and volunteered the information that the feeling of oppression over his left eye was gone. The deviation of the eyes was so far improved as to enable him to distinguish objects before him so clearly as to count fingers accurately and recognise faces. The deviation, however, was still considerable. This state of matters went on till June 23rd, when he became capable of voluntarily overcoming the deviation for a few moments, in the effort turning the eyes upwards as well as to the left, and parting pretty copiously with tears. Six weeks after the operation he could sustain this effort much longer, but so soon as the effort ceased, the eyes returned to the old position. He could get his way about without groping, and could see and distinguish objects very well. The following report, which has been kindly furnished me by my friend Dr. Little, of Manchester, gives a precise description of the boy's condition before and after operation.

"James Lovely was sent to me on May 15th, 1882, by Drs. Ross and Thomson, for ophthalmoscopic examination. The boy was led into my consulting-room by an attendant, and appeared to be quite blind.

"There was excessive deviation of the right eye, directly outwards, and of the left eye inwards; both were fixed in this position. The corneæ and pupils were almost completely hidden, and it was found impossible to make an ophthalmoscopic examination.

"So great was the deviation that I formed the opinion the contraction was due to some central irritation, and not to paralysis; besides, I had never seen a case of ocular paralysis in which the fundus of the eye could not be examined. I made no note on the condition of the pupils, or the perception of light.

"I next saw him on the 7th of March, 1883, and his condition was as follows:—

"*Right Eye.*—The muscular movements were normal—slight corneal opacity; pupil active; optic nerve was paler than normal, but of fairly good colour; its outline was well defined, with the exception of some faint irregularity at one side; the field of vision was slightly contracted on the temporal side (nasal portion of retina)—the vision was equal to $\frac{20}{30}$.

"*Left Eye.*—Muscular movements were almost equal to the right eye, the external rectus only was slightly weaker; pupil fixed, and no perception of light.

"The optic nerve was white and atrophied, and its outline was well defined."

Notes and Remarks by Dr. Ross.

May 15*th*, 1882.—James Lovely was sent to me for examination by my friend Dr. Thomson, of Oldham, and the following notes were taken:—

The patient is a very healthy, well-developed and intelligent boy. The depressed portion of bone corresponds to the infero-postero-parietal area of the skull on Turner's diagram, which would overlie the posterior part of the middle temporo-sphenoidal convolutions, and the inferior portion of the supra-marginal and angular gyri.

The patient cannot distinguish objects, but he is sensitive to the light of a taper placed immediately in front of the right eye. He soon loses even this glimmer when the taper is slightly moved in any direction. The sensitive portion of the retina is situated in the external or temporal half. The patient is totally blind of the left eye. Both eyes are drawn to the right, and slightly downwards, and the head is also slightly rotated to the same side, the attitude thus assumed making it appear as if the patient were endeavouring to look over his right shoulder. The attitude of the head seems to be in great measure produced by contraction of the right sterno-cleido-mastoid muscle, which is slightly more prominent than the corresponding muscle on the left. In addition to the conjugate deviation of the eyes to the right, slight nystagmus movements are observed in them. When the patient is asked to direct his eyes to the left, they are drawn still further to the right, and a similar action takes place when an attempt is made to open the eyelids in order to examine the eyes. Under these circumstances, indeed, the eyes are drawn so far to the right and downwards that the irides and even the corneæ become almost completely hidden, and this has rendered it quite impossible to conduct a successful ophthalmoscopic examination of the optic discs. The eyes appear to be much retracted, and the palpebral fissures are very narrow, and that this condition did not exist prior to the accident is rendered probable by a statement volunteered by the patient that his mother remarked that his eyes were now "much sunk in his head." The pupils are in a medium state of contraction, and both of them react to light. The lachrymal secretion is very abundant, and the patient usually holds a handkerchief in his hand, which he constantly uses in wiping his eyes. He states that he sometimes sees blue lights, but he has not experienced any of the hallucinations or illusions of sight. Taste, smell, and hearing are normal, the latter being particularly acute on both sides. The mental faculties seem to be entirely unaffected.

March 7th, 1883.—Several opportunities have been afforded me of observing the progress of this boy's case since the date of the operation, but there are only one or two circumstances connected with the case which it is necessary for me to mention. I saw the boy in July 1882, two months after the operation, and I then found that both pupils contracted when light was admitted to the right eye, but failed to react on light being admitted to the left eye. The left was considerably smaller than the right pupil, as it has since remained. In February of this year the boy became an inmate of the Barnes Convalescent Hospital at Cheadle; he was suffering on admission from photophobia, lachrymation and conjunctivitis of the right eye, along with a superficial corneal abrasion. The boy states that this was the third or fourth attack of similar character from which he suffered since his accident. Dr. Little's report renders it unnecessary for me to say anything further with regard to the movement of the eyeballs and the state of vision.

Remarks.—In experimental investigations on animals two areas have been discovered in the cortex of the brain, irritation of which gives rise to rotation of the eyes and of the head to the opposite side. One of these is situated in the posterior portions of the superior and middle frontal convolutions (No. 12, Ferrier's diagram), and the other occupies the supramarginal lobule and angular gyrus (No. 13, 13^1, Ferrier's diagram). Ferrier believes that the last area is the visual centre, but whether this interpretation be accepted or not the fact remains, that irritation of this area produces a conjugate deviation of the eyes and rotation of the head and neck, which are directed to the opposite side. Strong clinical evidence has been adduced by Grasset[1] to show that destructive lesions of the angular gyrus and supra-marginal lobule produce a conjugate deviation of the eyes and rotation of the head and neck, which are directed towards the side of the lesion. It was evident that the conjugate deviation of the eyes and the rotation of the head in this case was of the spasmodic variety, and it was most likely to be caused by a spiculum of the internal table of the skull, which was projecting inwards at the seat of injury, and thus maintaining the angular gyrus in a constant state of irritation. It was therefore confidently anticipated that, should the patient survive, the movements of the eyeballs would be much improved by an operation which would remove the projecting portion of bone. The cause of the defect of vision present in this case was much more difficult to unravel. It was evident there was complete blindness of the left eye, but a slight degree of vision still remained in the right eye. The patient saw a glimmer of light when a lighted taper was held in front of the right eye, and from the extreme rotation of the globe the rays of light must have fallen upon a peripheral portion of the temporal side of the retina. When the taper was moved horizontally outwards, so that the rays would fall upon the more central part of the retina, the patient failed to see the light. It was therefore evident that the loss of vision of the right eye was not due entirely to the extreme rotation of the eyeball. Two views suggested themselves as to the cause of the visual disorders. The first was that the loss of vision was caused by changes in the optic nerves and tracts set up by a basal meningitis. That there was basal injury was rendered probable by the presence of photophobia, lachrymation, and attacks of neuroparalytic opththalmia, all of them evidences of irritation of the fifth nerve or of the Gasserian ganglion. But the persistence of the pupillary reflex in the left or blind eye rendered this view doubtful. The second view with regard to the cause of the loss of vision was that it resulted from injury of the hemispheres of the brain, situated above the corpora quadrigemina, and thus above the reflex arc for the regulation of the movements of the pupils to light. The experiments of Ferrier and Yeo showed that ablation of both occipital lobes as far forwards as the angular gyri caused absolute and permanent blindness in the monkey, and from the nature of the injury in this case the occipital lobes could not fail to have been extensively damaged. If the second opinion were true, it gave grounds for hope that the removal of the depressed portion of bone would lead to a considerable improvement of the vision of the right eye, and a report of the case given by Drs. Thomson and Little shows that the most sanguine anticipations formed of the benefits likely to result from the operation have been fully realised. A symptom which I am unable to account for is that the pupil of the left or blind eye is considerably smaller than that of the right eye.

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[1] Grasset, 'Des localisations dans les maladies cérébrales,' Paris, 1880, p. 215, *et seq.*

Reviews and Notices of Books.

On Concussion of the Spine, Nervous Shock, and other obscure Injuries of the Nervous System, in their Clinical and Medico-legal Aspects. By JOHN ERIC ERICHSEN, F.R.S., &c. Longman, Green, & Co. London, 1882.

Injuries of the Spine and Spinal Cord without apparent mechanical Lesion and Nervous Shock, in their Surgical and Medico-legal Aspects. By HERBERT W. PAGE, M.A., M.C. Cantab., &c. J. and A. Churchill. London, 1883.

THESE two works, published within a short time of one another, treat of the same subject,—namely, injuries to the vertebral column and spinal cord resulting from railway accident, and the series of nervous symptoms which as a consequence ensue. This is a most difficult and important practical question, involving, as it does, on the one hand, the comfort and well-being of a large number of persons who are unfortunately the victims of the modern extension of steam progression, and on the other, the financial liabilities of the various companies who are called upon in these circumstances to compensate the sufferers. Finally, and this is by no means a consideration of little moment, the subject is one which greatly affects the credit of our own profession, which is of necessity intimately associated with most decisions in the matter; and there is no problem to be solved in medicine or surgery more obscure, which gives rise to more difference of opinion, or which more taxes the acumen and knowledge of the practitioner, than a just appreciation of the many complicated details in the diagnosis and prognosis of such cases. From want of accurate statistical and pathological data, and from the extreme complexity and uncertainty of all nervous phenomena, the conscientious medical man has always, in his scientific capacity, a difficult and delicate task in forming a sound conclusion; and as a judicial adviser, his embarrassment in weighing the scales of justice is further increased. In such circumstances, when powerful conflicting interests clash—when on the one hand there exists personal sympathy for the sufferer, and on the other a desire to guard against imposture—it is not surprising that diverse opinions should be advocated, and these not seldom in a manner little creditable to the medical profession, or calculated to facilitate the labours of the judge and jury engaged in arriving at a verdict. Such unfortunate conflicts will only be favourably modified by a more precise and comprehensive knowledge of the entire subject; and with the aid of modern improved methods of research, an extension of experience, and the scientific utilisation of accumulated facts, we may hope that this will be attained. The profession must therefore hail with satisfaction the recorded efforts of those whose opportunities have given them that experience on a large scale which falls to the lot of few, and whose conclusions are offered for the guidance of their less initiated, but sometimes equally responsible, brethren.

Mr. Erichsen's work is the revision of a former edition, published some years ago. The views therein contained are essentially the same as have been advocated for a long period by this author, whose experience and reputation as a surgeon are sufficient guarantees for the excellence and usefulness of the volume. To discuss in detail all the important features of the book would be here impossible; it is sufficient to say that the whole question is considered in a complete and masterly manner, indicating the great practical experience and thoughtful consideration of the writer. It is stated that the results of railway accident are not peculiar or characteristic, but that they only differ from other injuries in degree of severity. It is pointed out that blows on the back may give rise to local muscular, ligamentous, or nervous disturbances, or to fracture or other derangements of the vertebral column, with their accompanying symptoms. These last by pressure, hæmorrhage, or otherwise, may induce organic disease in the cord itself, of varying degrees and kinds. It is further urged that such accidents, whether the injury be direct or not, may by concussion of the spine give rise to a series of nervous phenomena attributable to alteration in function of the spinal cord, in which no organic changes in its substance appear to exist, but which can only be explained by molecular abnormality. Again it is asserted that, after such accidents, in certain cases, even when there is no local injury to the vertebral column or its contents, there arise in the cord secondary permanent changes of an inflammatory character as the result of slight or severe concussion. These are followed by degeneration of the nervous centres, and the sequence of symptoms usually met with in idiopathic diseases of the same nature. Such an occurrence must be looked upon as of the most serious import, and when advanced the prognosis of recovery is practically hopeless. The author admits, that it is not known how indirect concussion alone should lead to these inflammatory changes in the cord; but he assumes that these must exist, from the symptoms subsequently displayed by the patient. Finally, concussion may induce physical or mental shock, and the various phenomena resulting from fright and mental anxiety; the psychical disturbances being either functional or organic in character, the latter generally being represented by cerebral meningitis. The symptoms, pathology, treatment, and medico-legal aspects of all these points are submitted to careful investigation and discussion, for the details of which we must refer our readers to Mr. Erichsen's volume.

Mr. Page's work treats of the same subject, practically going over the same ground as the book to which we have just referred, and the conclusions arrived at represent the experience of nine years as medical adviser to a large railway company. This also will well repay careful perusal, consisting as it does of a thoughtful consideration of the whole subject, and the outcome of considerable practical experience. Although agreeing in the main points, in some important particulars the author dissents from the explanatory opinions of Mr. Erichsen, and a considerable portion of his work is devoted to a critical discussion of that writer's conclusions. Mr. Page maintains that the expression "concussion of the spine" is a misleading one, being ambiguous in meaning, and doubtful as to existence. After railway and other accidents he admits that the spinal cord may be injured and subsequently become diseased with corresponding symptoms, as a result of fracture or displacement of bone, hæmorrhage into the membranes, &c.; but he denies that concussion or shock alone can give rise to organic degeneration, or even to abnormal molecular alteration in the nerve-centres. The cord, from its position, it is urged, is protected from indirect impressions, and there is no evidence that the symptoms described are the results of inflammatory invasion, nor can such be proved by post-mortem examination. In short, Mr. Page maintains that the symptoms in the large majority of those indirect injuries following railway collision can be explained by strain or injury to the muscles, ligaments, and nerves of the back, to which may be added the effects of nervous shock and those moral influences resulting from fright and anxiety, and that they have no necessary connection with gross or irrecoverable diseases of the nerve-centres. The conclusion he arrives at is, that in favourable circumstances most of such cases may confidently look forward to recovery, and that even when the symptoms are apparently grave the prognosis is far from hopeless.

A superficial consideration of these two works would lead the reader to suppose, that the authors were seriously at variance with one another on the subject which they attempt to elucidate; but it seems to us that the discrepancy of opinion is more apparent than real, and that it is only in the explanation of certain phenomena that a difference in their views exists. Both agree that direct violence to the back may induce injury to the muscles, ligaments, bones, and nerves, of a more or less permanent character; and that these in turn, by mechanical pressure or otherwise, may be followed by inflammatory or other degenerations of the spinal cord of a gross and dangerous nature. Both, again, admit that after an accident, whether the injury be direct or indirect, symptoms of shock or other functional derangements of the nervous system may ensue without organic changes, the results of which may, on the one hand, be slight and temporary, or, on the other, severe and permanent. The main point of issue between the two authors is whether or not the general concussion or jar to the vertebral column is capable, without local injury, of so injuriously influencing the structure of the cord as to give rise to the severe nervous symptoms frequently induced. Mr. Erichsen agrees with Brodie, A. Cooper, Abercrombie, Bell, Ollivier, and many other authorities, in believing that this is the case, while Mr. Page maintains that there is no evidence whatever to show that organic, or even molecular, disturbance of a pathological nature in the nervous system results from mere general concussion or shock, and that the symptoms may be explained on other grounds.

A correct appreciation of this disputed point is obviously of the greatest practical and scientific importance, as the prognosis and consequent evidence in cases of litigation will depend upon the view thus adopted in each individual instance. The greatest possible difficulty has always existed in settling this question. The chronic nature of the complaints, the uncertain and protean character of all nervous affections, the tendency to exaggeration and deception on the part of the patient, the pecuniary and other interests involved, and, above all, the almost total absence of reliable pathological data, combine to embarrass the observer.

In favour of the view that general concussion may subsequently induce inflammation and other affections of the cord and its membranes, it may be urged that the symptoms which sometimes ensue after such accidents can only be explained on that supposition, as they accord with those we are accustomed to meet with in well-known idiopathic affections of a similar nature. The paralysis, rigidity, wasting, and loss of sensation would seem to be accounted for only by some permanent and definite change in the spinal centres. That such alterations should occur as the result of a violent jar is not surprising when the delicate and complicated structure of the cord is considered, and how readily its nutrition may be modified by external disturbances. Mr. Erichsen, in support of this view, cites a number of cases of general concussion from accident, without recorded, known, or discoverable local injury, in which the subsequent nervous symptoms were so severe and permanent as to induce him to diagnose inflammation of the cord and its membranes; and in the only case he had an opportunity of observing after death, cerebro-spinal meningitis was actually demonstrated. It may be admitted that there is little pathological evidence of this assertion, but the rarity of post-mortem examinations is not necessarily a proof that these conditions do not exist, but may be due to the special difficulties connected with such cases. The progress of the malady is generally extremely chronic, the patients rarely die for many years, and when they do, often from some complication, the original cause of their ailment has been forgotten in the prolonged misery of the secondary complaint which has been treated as a nervous disorder.

Opposed to this theory powerful arguments can be advanced, and it may be maintained that there is no proof whatever that the spinal cord is in any way injuriously influenced by indirect concussion, but, on the contrary, that the security of its anatomical position would seem to render this unlikely. Considering the frequency of railway accidents, if this induced organic affections of the cord, it may be urged, we should frequently meet with such in practice, and have opportunities of investigating their tissues after death. On the contrary, both these occurrences are rare, and the latter conspicuous by its absence. In the few cases where definite changes have been determined, there remains the doubt whether or not there was pre-existing disease unconnected with the alleged injury. Again, the vast majority of these railway cases ultimately recover, and Mr. Page cites a consecutive series of 234 instances, followed by more or less serious nervous symptoms, in only one of which was there any evidence that disease of the cord existed, most of the patients subsequently regaining their normal health. Further, it may be maintained that even when serious symptoms arise after a violent accident, it being admitted that the cord is diseased, when there is no examination after death we cannot be certain that the lesion is not the result of fracture or dislocation of the vertebræ, hæmorrhage into the membranes, or other local injuries to the nervous centres. There is also no evidence that the symptoms attributed to disease of the cord are so in reality, but there is reason to believe that they can be otherwise explained. For example, the pain and tenderness of the back so often complained of may be accounted for by strain of the muscles and ligaments rather than by affection of the cord itself, in the diseases of which this symptom is not necessary. In all nervous persons, and therefore most likely to occur in those suffering from fright or shock, neuralgic pain in the back is an almost universal phenomenon, even when there is no suspicion of central disorder. The symptom, therefore, of pain in the back gives us little information in such cases. The paresis, rigidity, modifications of sensation, psychical and other symptoms, may all occur as a result of fright and mental shock, and need have no necessary connection with organic disease of the spinal cord. In short, as Mr. Page contends, the statement that inflammatory action can be set up in the nervous centres by indirect concussion of the spine or vibratory jar is a pure hypothesis, and unsupported by clinical or pathological data.

To arrive at a sound conclusion in the face of this important difference of opinion is, for reasons which have already been alluded to, a matter of the greatest difficulty, and in the present state of our knowledge it may be said that the problem has not yet been solved. Apart from accident, our knowledge of the causation and nature of nervous diseases is attended with much uncertainty and doubt. During the last few years great advances have been made in neurological science, and conditions are now differentiated and anatomically displayed which at no distant date were absolutely unknown. Even now our knowledge is far from perfect, and it is not surprising that in the special department we are now discussing much confusion and variety of opinion should have arisen. This will only be dissipated by increased special research and extended general information. As far, however, as modern experience extends, it may be said in general terms that in many instances a too sombre view has been taken of the symptoms following railway shock, and that those conditions attributed to organic disease of the cord were in reality due to fright or mental influences of a recoverable nature. While admitting that this represents the majority of cases, are we justified in denying the possibility of indirect injury, shock, emotion, or even prolonged functional derangement, being the starting-point of organic disease? A discussion on this point necessarily leads us into the realms of hypothesis. Of the etiology of all so-called idiopathic organic lesions we practically know nothing, but there are many collateral circumstances which tend to throw light on the subject. For example, in the practice of the neurologist, in a certain number of cases of undoubted cord-disease, the patient attributes the origin of the affection to some slight fall or blow, which perhaps at the time was scarcely noticed. The experience of surgeons also can furnish examples, demonstrated by post-mortem examination, of abscess of the brain and other definite lesions of the nervous centres, the beginning of which could be traced to slight injuries, not local in character. It is of course impossible always absolutely to fix cause and effect; still the coincidence is of sufficient frequency to demand consideration. It may be that such collisions are only the exciting cause of disease in persons predisposed. In any case the association is obscure; but should the fact be admitted, we are forced to assume that the physical and mental disturbances are the result of nutrition changes caused by the shock, which ultimately lead to alteration in structure. The same applies to railway accidents, in which it is admitted that some cases are recorded in which inflammatory conditions of the cord were demonstrated after death, in circumstances which left little doubt that the collision was the starting-point of the disease. We ourselves have heard of at least one instance in which a man was treated and compensated for a supposed functional paralysis following a railway accident, whilst after death acute spinal meningitis was demonstrated.

Again we may ask, can chronic functional disorder lead to gross organic change? Although there is no actual proof, we have every reason to suppose that this may occur. For example, simple disuse of a muscle or organ is followed by atrophy, a visible condition showing functional arrest to be attended by tissue alteration. Recently it has been maintained that many of the system diseases of the cord, such as locomotor ataxy or lateral sclerosis, may derive their starting-point from perverted functional activity. There can also be adduced evidence to show that a disordered periphery may lead to central change, as cases are on record of disease of the centripetal nerves being followed by degeneration of the cord. Besides the possibility of functional or peripheral peculiarities being the active cause of permanent central changes, external impression alone may have the same effect. It is well known that loud noises or intense light may cause complete and permanent deafness and blindness, and such could scarcely exist without some ultimate form of tissue change. Even mental emotion, such as terror and fright, are known to induce definite objective phenomena; and it is not going far to assume that if the effects are permanent, they must be represented by structural alteration. If this is so, there is nothing unreasonable in asserting that the same may follow the bodily and mental shock, the result of a violent railway collision.

These considerations seem to illustrate the extreme complexity and obscurity of the question under consideration, and to suggest that we are not yet warranted in forming too dogmatic opinions on the subject. In the case of nervous symptoms after accident, it is probable that in the majority there is no evidence of central disease; but we are not justified, in exceptional cases, in denying its possibility.

There are many other points in the works before us which might be discussed with interest and profit, a consideration of which space compels us to forego. On one subject, however, we shall make a few remarks; namely, the value of electrical tests in the investigation of nervous affections. If we understand Mr. Erichsen rightly, he appears to believe that a relation exists between the amount of paralysis and the susceptibility to current stimulation. He says, "The loss of motor power in the foot and leg is best tested by the application of the galvanic current . . . . and a very true estimate can be made of the loss of contractility in any given set of muscles." On the other hand, Mr. Page rather depreciates the utility of this method of investigation, and considers that undue confidence has been placed in its efficacy—at least, when left, as it too often is, in the hands of incompetent observers.

In the first place, it may be stated that electrical reactions bear no relation whatever either to the amount or duration of paralysis, as this symptom may be complete and may have existed for years without any abnormal response either to nerve or muscle. We have seen cases of paralysis of thirty years' standing in which the responses to the currents were practically healthy. It is only in certain forms of paralysis that we meet with positive phenomena. The absence of these is therefore no proof that loss of voluntary motion does not exist, and this may be present to a great extent and for a prolonged period, with perfectly normal electrical conditions.

In the second place, it may be admitted that, in the large majority of cases of paralysis after railway accident, the electrical tests afford us little information, as the results are simply negative. This is not on account of any defect in the agency employed in their investigation, but of the fact that the class of diseases in which abnormalities in its action occur is in the circumstances a very small one. Such being the case, it is no less true that when positive phenomena are demonstrated, they are of the utmost value as sure indications of organic disease, and may be depended upon as implicitly as the morbid indications discovered by the stethoscope and thermometer.

It must, however, be stated that the practice of electrodiagnosis is one of the greatest delicacy, and can only be successfully carried out by those who have specially studied the subject in all its bearings, and made themselves masters of the manipulative details with which recent researches have supplied them. Nothing can be more unsatisfactory than the rough-and-ready method in which this process is usually practised; and as a result of the imperfect observation thus obtained, the most disastrous and erroneous deductions are frequently drawn. In the hands of the adept, electricity as a physical agent in diagnosis of diseases of the nervous system is assuming a position not second to optics and acoustics in the investigation of other affections. If from the nature of the paralysis, in most cases of railway accident, its phenomena happen to be negative, still, when they are positive, they afford the most reliable and definite information.

We have thus ventured to discuss some of the more important controversial questions treated of in the two works before us. We should not have done so, were we not convinced that the facts and conclusions of the authors were in the highest degree worthy of consideration and study.

A. HUGHES BENNETT.

De la Paralysie Générale et du Traumatisme dans leurs rapports réciproques. Par le Docteur CHARLES VALLON, Ancien interne de l'Asile Sainte-Anne, etc. etc. Paris: Delahaye et Lecrosnier. 1882.

IN this able and interesting monograph M. Vallon seeks to inquire into the influence which injuries, more especially blows and falls upon the head, have in causing the development, or in hastening the march, of general paralysis. He begins with a brief *résumé* of the opinions of authors who have written upon the subject, of whom a goodly number—Lasègue, Ball, Baillarger, Lunier, Voisin, Decorce, Azam, Skae, and others—have already recognised the important *rôle* played by injuries to the head in the etiology of this classical disease.

The inquiry undertaken by the author is an exceedingly difficult one, for there is perhaps no question more often insoluble in the life-history of disease than the time and cause of its beginning. More especially is this the case in the history of diseases of the nervous system, for the very subtlety and insidiousness of their origin render it well-nigh impossible to say when this or that malady really began. Who shall tell at what moment the degeneration of tabes attacked its favourite columns in the spinal cord, or when a general paralytic passed the boundary-line between health and disease? And if a long-continued functional disturbance may sometimes precede the outbreak of organic and incurable lesion of the nervous centres, as there are some grounds for believing, how doubly hard must it be to name with certainty the causes which contribute to or even induce the lesions which are at length made manifest by their peculiar symptoms! How easily in such circumstances may injury seem to be the starting-point of the disease, when it has in fact been nothing more than an agent in bringing to light those symptoms, which any concurrent illness or affection would have as surely revealed. The author is fully alive to these difficulties, and they seem to have been present to him in learning and recording the histories of the cases which he has himself observed. It appears to us open to question, however, whether the cases quoted from others have been investigated with the same impartiality and care, and whether they do not fail to provide us with unequivocal examples of general paralysis having origin in injury and in injury alone. There is nothing inherently impossible in the cerebral lesions which are frequently found in general paralysis being caused by severe falls or blows upon the head, in their being later results of the surface contusions which doubtless are often produced by so-called concussion of the brain; but many circumstances combine to show that the origin of this disease therefrom must at the most be excessively rare. It is a noteworthy fact that general paralysis of supposed traumatic origin is only to be met with at that period of life when the disease is ordinarily seen, and that the malady thus induced differs in no respect from that whose origin is seemingly more obscure. The author indeed admits that in those cases where the symptoms have appeared within a very short time of the injury, the injury cannot have been the sole agent in the induction of the disease, but that it has exercised its influence on a nervous system already predisposed to, or in imminent peril of, morbid attack. And if this be so, it must be still more difficult to trace the connection between the developed disease and some injury which has preceded it by a considerable lapse of time. In this category are cases, on the one hand, where the injury is followed by a continuous train of cerebral symptoms, due presumably to chronic hyperæmia—in the absence, we may fairly say, of any more certain pathology—and ending in a diffuse meningo-cerebritis. In such cases, the relation of cause and effect between the injury and the disease may forsooth be accepted as proven, if with certainty it can be said that no symptoms whatever existed before the accident, and that the fall or blow upon the head was in no wise due to a vertiginous attack, one of the earliest symptoms of the disease. On the other hand are cases in the same category where disease and injury are not linked by any chain of symptoms, and where the difficulty of tracing the connection between them must be almost insuperable, even if some mental bias be not necessary to see any connection at all. In the presence of that uncertainty which, with our present knowledge, must obviously exist as to the traumatic origin of general paralysis, it seems hardly prudent to assume—for an assumption it can only be—that in the cases last referred to, the injury to the head must have produced a commotion of the cerebral mass which predisposed to the origin and development of general paralysis, even though the symptoms of brain concussion have long since passed away. M. Vallon quotes from various authors, MM. Azam and Lasègue especially, who seem to hold this opinion, and to see in every severe concussion of the brain a danger from the jar, commotion, or *ébranlement* which the head received. It is this *ébranlement* which has been made so much of by some writers as a source of danger to the unfortunate victims of railway collisions,—that jar which in the absence of scratch or mark upon the body may nevertheless be the starting-point of a series of nervous phenomena, supposed to end in meningo-myelitis of the brain and spinal cord. Sufficient time has now elapsed, and there has been more than a sufficient number of subjects of this *ébranlement*, for this theory to be established, if there be any truth in it, on clinical and pathological data free from all question or reproach; and it is only adding a new terror to life if every blow upon the head which causes concussion of the brain is to entail a grave prospective risk for the sufferer in consequence of the molecular disturbance which his brain sustained. Concussion of the brain is one of the commonest injuries in railway accidents, but is there any evidence to show that general paralysis is even rarely induced by this special form of vibratory jar? We need, it seems to us, some clearer evidence than even in this able work the author has been able to adduce, of the undoubted origin of general paralysis from injury, before we can fairly regard molecular disturbance of the cerebral mass as a probable cause of the remote development of this disease. Life indeed would hardly be worth living if the doctrine were clearly established, that even the man who perfectly recovers from a severe blow upon the head is ever sitting—as one author puts it—upon a volcano which may burst out as general paralysis at any moment, the outbreak causing no more surprise than an eruption of Vesuvius even after a quiescence of many years. We refer to this part of M. Vallon's work more especially, because amongst the practical conclusions which he draws from a consideration of the whole subject is the medico-legal question, how far a blow upon the head in a railway accident may predispose the sufferer to general paralysis at some future time. He dwells with becoming force on the many errors which are prone to creep in unawares in the investigation of the origin of this disease; but let us pause before we raise this form of *ébranlement* of the brain to the same dignity as the supposed *ébranlement* of the spinal cord, with its inevitable though distant risks, and before we build, on unsure foundations, another theory to cause untold misery from the fear of coming evil, and incalculable injustice from the use which may be made of it.

In the second part of his work, M. Vallon deals with the influence of general paralysis on the course of surgical affections, such as fractures and wounds, and the influence which these in their turn have on the course of general paralysis. He points out how in the advanced disease the bones are in some cases so structurally altered as to be liable to fracture, and how in others there seems to be a great tendency towards suppuration and gangrene after the infliction of wounds. Considering, however, that the wounds of general paralytics have in many cases healed perfectly, and even by first intention, some evidence should surely be given as to the sanitary conditions and the treatment of wounds in the asylums of Paris, before we can admit that there is any special liability to suppuration, erysipelas, or gangrene, in the course of this disease. Were this really so, it is hard to believe that some authorities would go so far as to recommend the use of setons and issues as a means of alleviating or arresting the symptoms of general paralysis.

The monograph is closed with a series of thirty cases, of which several were observed by the author himself and his friends, and have not previously been published. To these we naturally turn for support and confirmation of the doctrines laid down, but we cannot refrain from expressing doubt as to the propriety of the heading which introduces many of them: "General paralysis of traumatic origin." Some occurred years ago; others seem defective in information as to the conditions of the patients before the alleged injuries; and most are strangely wanting in the record of post-mortem examinations. In the single case, moreover, where the post-mortem appearances are related, a case under the author's own observation and investigated with his wonted care, there is a hiatus—not unnoticed by himself—of nearly three years in the history, large enough to throw serious doubt upon it, even if the facts as they stand were adequate to establish it as an unquestionable example of general paralysis with a solely traumatic origin. The whole treatise, however, and the cases contained in it, are worthy of perusal, calling renewed attention as they do to the possibly traumatic origin of this terrible disease. For if the traumatic origin be well-grounded, there is better hope than heretofore, as the author points out in his conclusions, both in the clinical history and in the treatment of this hitherto hopeless malady. By a careful avoidance of all those conditions which aggravate or prolong the after-symptoms of brain-concussion, the onset of general paralysis from injury may be altogether prevented; and in those cases where the disease has unquestionably been caused by injury, the children born before the accident are free from the inheritance of a taint the knowledge of which can only be a source of life-long anxiety, largely conducing perhaps to the outbreak of the disease. HERBERT W. PAGE.

The Factors of the Unsound Mind and the Plea of Insanity.

By WILLIAM A. GUY, M.D., F.R.C.P., F.R.S.

THE publication of Dr. Guy's work on the Factors of the Unsound Mind, with special reference to the Plea of Insanity in Criminal Cases, is peculiarly opportune at a time when the codification of the criminal law is in contemplation. The special advantages which Dr. Guy has enjoyed for studying those forms of insanity which are most frequently associated with crime, entitle his views upon this subject to thoughtful consideration; while the reasonableness, moderation, and logical consistency with which these views are set forth, are likely to secure for them a favourable reception by the lawyers and legislators, to whom we must look for any amendment of the law. Dr. Guy is strictly scientific in his method, but he avoids those technicalities and pedantries that darken knowledge, and succeeds in making even abstruse topics clear and inviting. Deeply learned in the history of medical psychology, and possessing great literary skill, he has produced a treatise which is interesting and instructive to the ordinary reader, and which contains suggestions which may be useful to those who have made insanity their life-study.

The first factor of the unsound mind of which Dr. Guy treats is illusion; and from an able analysis of the most remarkable recorded cases of this affection, he concludes that illusions are brain-created sensations or revivals of past sense experiences, and are not necessarily connected with the activity of any faculty of imagination or organ of sense. Passing next to delusions, which he defines as involuntary thoughts, ideas, or beliefs, without data or premises, he classifies them into six groups, according to their nature and the sources from which they proceed, and then by an easy transition he shows how illusions and delusions are combined in dreams, which have long been regarded as an analogue of insanity. After dreams proper, somnambulism or acted dreams, and artificial somnambulism or mesmerism in all its varieties, are passed in review, and then comes a masterly summary of delirium, febrile and toxic, of incoherent speech, catalepsy, epilepsy, and hysteria. The three final sections of the first part are devoted to the consideration of the emotions, passions, and movements of the will; of insanity arising out of causes of common occurrence; and of the narrative of Mr. Perceval, which is used to illustrate the statements made as to the factors entering into the constitution of the unsound mind.

The second part of Dr. Guy's work, if not the most interesting, is certainly the most important and valuable, for in it we have the practical application of the principles laid down to the purposes of jurisprudence. The insane are divided into three classes—those who suffer from undeveloped, from degenerate, and from disordered mind; and the responsibility in courts of law of members of those several classes who have committed acts of fraud or violence is considered. The differential diagnosis of homicidal acts by imbeciles, by maniacs, and by patients labouring under insane impulse, is very carefully worked out, and it is satisfactory to find that Dr. Guy has no doubt as to the existence of sudden and irresistible impulse leading to violations of the law. The theory that it is justifiable to punish madmen, and that it cannot be considered unjust or rigorous to inflict the penalty of death on a mischievous being divested of all the perceptions of reason and humanity, is disposed of in a very convincing manner, and figures are quoted to prove that neither to the sane nor to the insane class among our criminals does the prospect of long imprisonment or detention for life in a lunatic asylum offer any attraction or temptation; while the punishment of death seems as if it might exercise a certain attraction or fascination. It appears that the execution of a lunatic murderer has almost invariably been followed by an increased crop of lunatic murders.

The amendment of the law which Dr. Guy, in common with all eminent medical jurists, chiefly desires, is the abolition of the knowledge of right and wrong as a test of legal responsibility. Quoting from the amended Criminal Code Bill, that "To establish a defence on the ground of insanity it must be proved that the offender was at the time when he committed the act labouring under natural imbecility or disease of or affecting the mind, to such an extent as to be incapable of appreciating the nature and quality of the act or that the act was wrong," he confesses that, like the late Lord Chief Justice, he cannot understand the definition, and he goes on to indicate the several classes of cases on behalf of which the plea of insanity must sooner or later be set up. These are—1, Cases of acute instinctive or impulsive insanity; 2, Cases of chronic instinctive or impulsive insanity; 3, Cases of epilepsy and homicidal impulse; 4, Cases of imbecility; 5, Cases of mania. In all cases of all these classes there is weakening or destruction of the power of the will. Dr. Guy would dispense with all attempts to ascertain the state of mind of the accused at the time of the act, with all questions bearing on his knowledge of right and wrong, legal and illegal, and his appreciation of the nature and quality of the act, whatever that may mean, and concentrate the attention of the Court on the one plain question,—Is the accused of unsound mind, and was he so about the time when the act was committed? He would also no longer object to the plea of insanity being set up in difficult cases of moral insanity and instinctive mania, the existence of which it is impossible to ignore. He would have a medical assessor present at every trial at which the plea of insanity is set up, to hear all the evidence and assist the Court; and he would have skilled witnesses selected by the College of Physicians or some scientific body, and not by the prosecution and defence.

On the question of corporal punishment in prisons, Dr. Guy's views, which are freely and confidently expressed, are open, we think, to grave objections. The punishment of pain, surrounded with proper restrictions and patiently held in reserve, is, he argues, eminently humane, just to the well-conducted, and very often the only instrument of reformation to the habitual offender. But exactly the same arguments which Dr. Guy now advances in favour of the retention of corporal punishment in prisons have been urged in support of its continued employment under various other conditions of life, and have been again and again refuted by the practical results of its abandonment. Dr. Guy seems to think that the lash is peculiarly advantageous in dealing with strange epidemics, of self-mutilation and attempts at suicide, which he has witnessed in prisons; but surely the fact that he speaks of such outbreaks of offences as epidemics betrays his own recognition of a morbid element underlying them, and surely the arguments which he has himself marshalled against the punishment of the insane, in relation to the penalty of death, may be equally adduced against their punishment by flogging. It is incontestable that lunatics do find their way into prisons of all classes, where their mental disease often remains long undetected; and it is obvious that the intractability, irregularities of conduct, and degraded habits which arise out of their diseased state, must render them singularly liable to corporal punishment, where that is retained as an instrument of discipline. There are good grounds for believing that lunatics—and lunatics, too, labouring under mortal disease of the brain,—have been flogged in prisons in this country, at no very distant date, for mere infirmities of body, which have been mistaken for vicious habits. The possibility of such an occurrence, of a being already burdened with one of the most terrible of human afflictions being subjected to this punishment, or of what is practically a dying man being tied to the triangle and flogged, is almost of itself sufficient to justify the abolition of corporal punishment in prisons, unless very strong countervailing advantages can be shown to attend its use. But with reference to the epidemics of self-mutilation and suicide in which Dr. Guy thinks corporal punishment so beneficial, and even essential, it is to be borne in mind that similar epidemics are being dealt with daily, and most successfully, in all our lunatic hospitals, without any resort to the lash. The uniform experience of those hospitals in which are congregated together tens of thousands of men and women, more violent, reckless, and unmanageable than are to be found in prisons, is that severity is subversive of true discipline, and that humanity and firmness will secure order and obedience where harsh measures would fail lamentably to do so. There is not an asylum office in any civilised country who would wish to return to the floggings which were at one time a regular branch of treatment in all institutions for the insane, and which were defended with arguments even stronger than those now advanced in defence of prison-floggings, for it was maintained that they were not only necessary for the preservation of order, but positively curative in their effects.

J. Crichton-Browne.

De l'Ataxie locomotrice d'origine syphilitique. Par A. FOURNIER, Professeur à la Faculté de Paris. 8vo. pp. 280. Paris, 1882.

SINCE the publication of his first statistics, Prof. Fournier has had under his observation 103 cases of tabes, in 94 of which he discovered syphilitic antecedents. His percentage, therefore, closely approximates that of Erb; the mean between the numbers given by these two observers being as high as 90 per cent. It is agreed by those who uphold the syphilitic causation that tabes is a tertiary manifestation of the disease; and our author further shows that out of 89 cases, the first symptoms made their appearance from 5 to 12 years after the alleged infection in 55 cases.

With reference to the special causes determining the localisation of the syphilitic processes in the spinal cord, Prof. Fournier has not discovered any in the majority of his cases, though in a few, heredity or "surmenage nerveux," that is, a kind of life leading to excess of nervous expenditure, had an undoubted share in the results. A fact of great importance is that in the immense majority of cases it was found that the original specific symptoms have been mild, or very mild. In 10 out of 84 cases only had they been "moderate," in none "severe." In this particular, therefore, syphilitic tabes seems to form no exception to the rule which obtains among the undoubted forms of syphilitic neuropathies. At the same time, what the author calls mild cases might be contested by others as being syphilitic at all. Another fact forcibly insisted upon by Prof. Fournier is that in almost every case of tabes with specific antecedents, the latter have not been treated with sufficient energy and perseverance. The duration of the treatment in 73 out of 79 cases did not exceed one year (in 46 of these it was under three to four months); and the measures adopted were often of the most insignificant kind.

The part of the book which describes the clinical aspect of syphilitic tabes offers many points of interest. It is obvious that from Prof. Fournier's definition of ataxy as a disease of specific origin, we cannot expect the author's description to differ materially from those given by previous observers.

His account of the initial, or "præ-ataxic" stage of the disease, will, however, be read with great profit and interest; and many curious facts are recorded under the heading "Associated forms."

The symptoms of the præ-ataxic stage are arranged in eight groups (sensory; oculo-motor; genito-urinary; locomotor; visual; gastric; laryngeal; various rare forms); its most striking feature—its polymorphism—is in a certain measure consonant with the known characteristics of syphilis. It must not be forgotten, especially in deriving therapeutical conclusions from the alleged results of certain modes of treatment in arresting the disease, that the progress and duration of the phenomena constituting this stage of tabes are essentially variable. Indeed these symptoms may constitute for years the whole of the disease, and spontaneous temporary arrèsts or retrocessions are by no means uncommon.

It would be a great mistake to imagine that the classical "lightning pains" have the fulgurating character from the beginning. They often make their appearance as sensations of various nature, and it is the duty of the physician carefully to inquire into the existence of such sensations, which are readily overlooked by the patients. Occurring in an old syphilitic subject, they are of grave omen, and indicate an immediate and energetic medication.

After discussing tabetic hyperæsthesiæ and anæsthesiæ, the author comes to pupillary and oculo-motor disturbances: hemiopia, strabismus, myosis, mydriasis, ptosis, and more extensive paralysis of the third pair—such is the order of frequency in which they have occurred in his experience. These symptoms often appear suddenly, and may disappear suddenly also; but recidives are then frequent. Prof. Fournier very justly complains of the ignorance which still prevails among medical practitioners concerning the symptomatic importance of these ocular disturbances.

Concerning the genito-urinary organs, the abnormal excitation noticed by Trousseau is exceptional; depression, both of desire and power, is the rule. Micturition becomes lazy. A very important symptom is the sudden and unconscious escape of a few drops of urine (and, in certain cases, of fæcal matter). Various other modes of vesical disturbance may become developed, slowly as a rule, and definitively; though cases of sudden onslaught and retrogression are not rare.

Too little attention is usually paid to the paretic symptoms of locomotion which precede true ataxy. Sensations of weight, loss of endurance, are common, though transitory, phenomena. At the same time motor uncertainty in the dark may be noticed. Visual symptoms are not uncommon, and usually of slow development; thus amblyopia and narrowing of the visual field may precede by many years the development of ataxia. Dyschromatopsia also may occur. Like oculo-motor disturbances, they are unfortunately too often overlooked in their ominous significance. The classical "gastric crises" may be represented by vomiting, or by gastralgia alone. Professor Fournier has also observed a case in which flatulency and violent gaseous eructations took their place. He classifies, with other authors, laryngeal crises under the headings of spasmodic cough, suffocative fits, and apoplectiform apnœa. A case of Krishaber's is related in which tracheotomy apparently saved the patient's life. A number of other symptoms are occasionally observed in the præ-ataxic stage, according to the author; some of so grave a nature, and so far removed from the typical tabetic manifestations, that it may fairly be asked whether they belong to the disease itself. We must watch lest the natural polymorphism of tabes should become a pretext for manufacturing "complex types" of the disease out of casual coincidences of phenomena not essentially connected with it, or setting up as tabetic, cases in which the typical posterior sclerosis forms but a member of a more generalised process implicating the nerve centres.

The transition from the first to the true ataxic stage may occur at any time from a few months to many years after the onslaught. It is not usually sudden, but very gradual. Ataxy must be looked for carefully. Professor Fournier describes minutely the tests by which it may be demonstrated. 1. The patient is made to walk to command, on rising from a sitting posture, to stop and turn round suddenly. 2. He is made to go downstairs. 3. Standing the feet together and the eyes closed. 4. Standing on one leg with or without the eyes closed. Patients are often astonished, when made to get through these performances, to find how much they are deficient in equilibration; their ataxia had failed to be noticed by themselves.

The author next mentions the loss of the knee-jerk, which it seems to us ought to have figured among the very earliest symptoms of the præ-ataxic stage. His account of it is very deficient. A case has come to our knowledge which shows that the loss of tendino-muscular reaction may precede ataxy by very many years. When in such a case the syphilitic infection has occurred after this loss, are we justified in classifying it as one of "specific tabes"? This fallacy is patent, and yet it does not seem to have been duly taken into account in the statistics collected to prove the syphilitic nature of tabes.

We briefly pass over the description of the ataxic period, in which the author recognises several forms: amaurotic, articular, hemiataxic. He dwells chiefly upon the "associated" forms, which comprise ataxy with spinal (paraplegic and atrophic), and with cerebral complications.

Cerebral phenomena are met with during the præ-ataxic stage in the shape of psychical disturbance, epileptiform and apoplectic seizures, hemiplegia, and the pseudo-general paralysis of the author. Such cases, however, offer wide departures from the normal tabetic type.

Similar phenomena more frequently accompany tabes in the latter stage of its evolution. True, general paralysis has long been recognised by various authors as an epilogue of tabes. Now, for Professor Fournier, all such complex cases, which he calls "posterior cerebro-spinal syphilosis," are particularly characteristic of ataxy of specific origin; but would it not be more rational to consider some, at least, as cases of more diffuse sclerosis in which the posterior columns are involved? It is certainly difficult to draw the limit where to stop; is it scientific, however, to call "progressive locomotor ataxy" every case where those columns become the seat of morbid changes?

The question of antisyphilitic treatment in tabes is important, and the author is very explicit in his statements concerning it. He refuses to consider as treatment the half-measures too frequently adopted. In all specific diseases of the nerve-centres a most energetic, persevering, and repeated exhibition of mercury and iodide is indicated. He alternates these drugs every three weeks. The doses of iodide must be from one to two drachms per diem; of mercury, as perchloride, half a grain to nearly one grain, or, as strong ointment, from two to five drachms daily. Such courses are to be resumed from time to time during some years, in view of the otherwise inevitable relapses. Professor Fournier does not say he has ever cured a confirmed ataxic. He holds this to be impossible, owing to the actual destruction of tissue which has taken place; but he has seen such cases where certain symptoms improved under treatment, or even where the progressive invasion appeared suspended for a more or less considerable period. It is not impossible that, when obtained at an earlier stage, this arrest might prove permanent; but many years must have elapsed before one has any right to assume this to be the case.

The objections raised against the specific nature of tabes are next considered by the author. The chief ones are that — 1. Syphilitic tabes has no special symptoms of its own; to which it may be answered that, given an organ of which the functions are interfered with, by whatever cause, the visible results shall be the same. 2. It has no lesions of its own. Naturally enough, since the connective tissue which constitutes the sclerotic change does not histologically differ, to whatever cause be due its proliferation. 3. Syphilis does not produce systematic lesions. This is an *a priori* assertion, retorts the author—and besides, is tabes a strictly systematic disease? Does it not involve other parts, besides the posterior columns? And does not syphilis occur in certain subjects as "systematised" in the bones, or in the lymphatics, or in the mucous membrane?

4. Tabes is not cured by a specific treatment. This may be due to the fact that such a treatment is usually instituted too late; cases of necropsy show that extensive organic lesions may exist before the symptoms are at all well marked. And why should we expect to restore a sclerosed cord more readily than a sclerosed testicle, by mercury and iodide?

5. The connection between syphilis and ataxy is one not causal, but coincidental. What rational observer will be prepared to say this after the late statistics? Nor does syphilis

act as a merely "predisposing" cause to tabes, more than it does to the universally recognised cerebral syphiloses, affirms Professor Fournier, who, however, does not meet the obvious rejoinder that a respectable minority of cases of tabes are devoid of syphilitic antecedents, whilst the lesions of cerebral syphiloses are of a very definite type, not imitated by non-specific alterations. But as he strongly insists upon the frequency of "ignored infections," we may reasonably assume that he is not far from the opinion that all cases of tabes are of specific origin—an extreme, but at any rate consistent, view. We should like, however, to hear what the author has to answer to the objection that, whilst the other manifestations of syphilis are no respecter of sexes, tabes is of such rare occurrence among women. But he does not even allude to this important aspect of the problem.

The author, finally, passes in review the reasons which plead in favour of a true syphilitic tabes. First comes the recognised frequency of specific antecedents. Next, the fact of tabes occurring usually in the tertiary period, and its associations with nervous accidents, and other lesions common in syphilis. Again, the effects of an active exhibition of mercury and iodide. Lastly, the difficulty of finding a cause, other than syphilis, to account for the disease. All these points are shortly but vigorously handled, and lead to the general conclusion that our only hope in dealing with tabes is to seize it at the very earliest stage, and then, upon the least evidence of pre-existing syphilis, to follow the energetic plan of treatment above described.

Professor Fournier's volume is exceedingly well written. Here and there his enthusiasm for his theme makes him rise almost to eloquence. His arguments, based upon an immense experience of syphilis in its Protean manifestations, are worthy of the respectful consideration of the reader. But it must be admitted that, from the neurologist's point of view, his grasp of the subject before us is not so ample and steady as to stifle every doubt. So pleasant, however, is the perusal of his lectures, that we feel sorry not to have been more than half convinced by his able argumentation.

A. DE WATTEVILLE.

Critical Digests and Abstracts from Journals.

THOMSEN'S DISEASE.—*Tonic spasms in Voluntary Muscles. Muscular Spasm at the commencement of Voluntary Movements. Hypertrophic Spasmodic Spinal Paralysis.*

BY PAUL M. CHAPMAN, M.D.

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The disease of which some account is here given does not appear to have attracted the notice of English authors, a fact which may be due in part to its extreme rarity, and perhaps also to the fact that the only text-book in which it is described (Erb's volume in 'Ziemssen's Cyclopaedia') is practically out of the reach of most readers in this country.

A passage occurring in Sir Charles Bell's work, 'The Nervous System of the Human Body,' first published in England in 1830, and a few years later translated into German, has much attracted the notice of the authors who have written on this subject. The paragraph is slightly misconstrued in the German translation with which the authors are familiar. The original is headed 'Affections of the Voluntary Nerves,' and is here quoted at length:—

"I could give cases of various affections of the voluntary nerves, but the patients might be made uncomfortable by a report of their condition. The most common instance is an impediment in speech, when the consent of the muscles is imperfect; but this sometimes extends to all the voluntary muscles of the body. I find that some are capable of lifting a heavy weight, or walking fifteen or twenty miles, and yet they have not the proper command of their limbs; there is an insecurity and want of confidence in the motions of the body, which overtakes them upon any excitement; a paralysis of the knees which prevents the individual from putting one leg before the other, and which endangers his falling. Thus, a gentleman capable of great bodily exertion, on going to hand a lady to the dining-room, will stagger like a drunken man; and in the streets any sudden noise, or occasion of getting quickly out of the way, will cause him to fall down, and in this manner a want of confidence produces a nervous excitement which increases the evil. With confidence, the power of volition acts sufficiently; there is neither defect of speech, nor irresolution in the motion of the limbs, when the person is at ease or under a flow of spirits. Such cases are very curious in their details, as exhibiting an extraordinary degree of incapacity for the affairs of life proceeding from slight defects. There is neither disease of mind nor of bodily organs; the corporeal frame is perfect; the nerves and muscles are capable of their functions and proper adjustments; the defect is in the imperfect exercise of the will, or in that secondary influence which the brain has over the relations established in the body."

It will be observed that in the above description there is no suggestion of any *rigidity* or *muscular spasm*, coming on at the beginning of any series of movements, and passing off when the same movement is frequently repeated. The following description, given by Peters, of the behaviour of a patient, suffering from what is now known as Thomsen's disease, will serve by way of contrast:—

"If he has been for some little time at rest, he has to use great efforts before he can succeed in undertaking a voluntary movement. This phenomenon is specially remarked in attempting to walk, . . . at first he remains firmly rooted to the ground, notwithstanding a visible effort, so much the more if he knows he is being watched. At last he succeeds in drawing first one and then the other leg forward, . . . gradually he loses his stiffness and awkwardness, and at last his walk cannot be distinguished from that of a healthy person."

This stiffness, in the words of Leyden, is "characterised by the fact that the voluntary muscles do not readily obey the will, and that intentional movements are arrested halfway, the muscles remaining in a state of tetanic rigidity."

There would be no need to proceed further to show that, with regard to the kind of cases to which Sir Charles Bell refers, it is by no means clear that he had to do with Thomsen's disease. It is true that some cases of stammering, accompanied with initial laryngeal spasm, have a close and interesting analogy with the affection to be presently described; but all such cases are very different from those arising from what is known as nervousness or diffidence, accompanied with, and increased by, a painful knowledge of the defect—such as are those alluded to by Bell.

A case related by MM. Ballet and Marie will be presented in detail.

M. S——, born in Cairo, came to Paris to consult Prof. Charcot. He asserted that he had only perceived the first symptoms of his affection about the age of 10 years. At this time he found he was more clumsy than other children, that he was inferior to them in bodily exercises, and that after sitting down he had some difficulty in rising. He perceived no difference in his arms before his 15th year, and it was only at the age of 17 that his affection began seriously to trouble him.

When about 12–15 years old he noticed that when he looked upwards his eyes became, as it were, fixed in that position, and, for the space of a second or two, he had great difficulty in bringing them to the horizontal. [This phenomenon disappeared about his 24th year.] At the same time (12–15) on turning his head to one side, it often became fixed in that attitude for some moments, owing to a stiffness of the muscles of the neck. There was also a hindrance to the movements of the tongue, only coming on at intervals, and obliging him sometimes to wait a moment or two to avoid stuttering. At times also he had the sensation of an obstacle at the level of the larynx, impeding the emission of sounds, with a feeling of constriction at this level (possibly a spasmodic contraction of the muscles of the larynx, analogous to that of the muscles of the body). He had never suffered from troubles of defæcation or micturition. The muscles of the face had never been affected.

Actual state.—When the patient wishes to make a movement, when a group of muscles enter into action, tetanic rigidity nearly always supervenes, a contraction of the muscles, lasting for one, two, or three seconds, accompanied by a painful feeling, the muscles being hard to the touch and standing out in relief. This takes place only at the commencement of any movement; when it is repeated for a certain number of times, and the limbs "se sont échauffés," it no longer occurs. Thus, in mounting a staircase, the patient at first feels a stiffness in raising his legs, then, after seven or eight steps all stiffness disappears, and he can ascend like an ordinary individual. It is the same with the upper limbs; when the hand is closed he remains for some moments without the power to open it. This leads to bizarre situations. In getting on horseback, when attempting to engage the foot in the stirrup, the left leg is seized with the spasm; when this has ceased, it is the right leg which is fixed in extension over the crupper; finally all disappears, and he can place himself comfortably in the saddle.

This transitory tetanic rigidity is diminished during digestion. Is augmented by fatigue, emotion, vexation, and cold; also by lying on the back, and abstinence from coitus for eight or nine days (patient's own statement).

The muscles are very well developed, though one could not say they were hypertrophied: their consistence normal in repose; pressure causes pain, they are more sensible to pressure than normal. The phenomenon of idio-muscular excitability (myœdema) is not presented by the biceps, but is distinctly obtained on the calf, in a slight degree on the triceps of the thigh. Passive movements can be executed with great facility, and one cannot provoke tetanic rigidity by this means. Tactile sensibility, and tendinous reflexes, normal.

An electrical examination was conducted by Dr. Vigouroux, who states that:

1. The muscles and nerves are less excitable, above all less isolable [this fact being probably due to some physical cause affecting the diffusion of the current] than normal; farado-galvanic irritability below the mean.

2. There is no variation from the normal formula, except that for some muscles the anodic contraction is too easily obtained in comparison with the kathodic.

3. There is complete absence of break contraction.

4. The dominant fact is the facility with which the contraction provoked by the current becomes tetanic, diffusion of the current developing a spasm of the neighbouring muscles.

5. This persistence is produced still better by faradisation of a nerve-trunk.

The authors have obtained a myographic tracing by which they establish the duration of the muscular spasm excited by the voluntary efforts to be 5½ seconds, which closely corresponds to the time given by others (Seeligmüller 5 seconds); the spasm was not shown when the experiment was several times repeated.

Bernhardt states that the electrical reaction conforms to the ordinary formula, and notes the following particulars: the contraction persists a little after the cessation of electrical excitation; during the flow of a faradic current a firm and durable tetanic contraction is not obtained, but undulations are produced which swell in one point to smooth out again and reappear a little further off. Percussion with Skoda's mallet produces the same effect as electrical excitation of short duration. The sensibility is intact.

Vizioli also remarks that the induced current gives energetic contractions which last some seconds, although the stimulus be removed, there being nothing noticeable with the galvanic current.

The patients appear to have been, in all the cases collected, of good muscular development; and, in many, to have suffered from great muscular hypertrophy (Seeligmüller (3 cases), Bernhardt, Vizioli, &c.). Bernhardt points out that the hypertrophy is symmetrical. In his first case he at first thought he had to do with a case of pseudo-muscular hypertrophy; but it can in no way be confounded with the latter disease, in which the muscles are extremely weak, some completely atrophied; in all cases the peculiar spasm being absent. Portions of muscle have been excised and microscopically examined by Ponfick, Petrone, Jacuziel and Grawitz, but show nothing abnormal.

Speech is by no means always affected, though it has been frequently observed to be so. In Bernhardt's case of a student-of-law, the patient had sometimes great difficulty in opening his mouth, with stiffness of the muscles both of jaw and mouth, "the movements of the tongue are not executed in every direction as easily as in a healthy person; speech is in no wise embarrassed." In this case there was enormous muscular hypertrophy, and there was—a noticeable point—*lordosis* of the lumbar vertebræ, as in two cases observed by Seeligmüller. This, however, is mostly absent, as in the case of M. S—, and may be more apparent than real. Bernhardt remarks that the psychical state was normal; it appears, however, that, in the majority of cases, the patient is of irritable nature, suspicious, reserved, occasionally suffering from attacks of giddiness, &c. Thomsen, himself a sufferer from the disease, says: "Bell errs when he asserts that 'there is neither disease of mind nor of bodily organs'—disease is there with all its symptoms." Bell indeed remarks that "the patients might be made uncomfortable by a report of their condition;" that the patients have a reluctance to hold any communication with any one concerning their condition is, by Thomsen and others, elected into a veritable symptom.

The disease has been caused in two cases by fright, arising from fire, at the age of 7 (Peters), and from robbers (Benedikt). Also by a fall from a carriage at the age of 5 (Bernhardt). In neither of these cases was there any suspicion of heredity. But the most interesting points in the etiology of the disease are its presence in several members of one family, its occasional alternation with mental troubles, and its almost constant appearance in early infancy. In Leyden's case a brother was affected; in a case of Seeligmüller's, one sister; in that of Strümpell, two brothers. In Bernhardt's case an uncle suffered from epilepsy.

Dr. J. Thomsen, of Kappeln, Schleswig, himself a sufferer for over 60 years, describes, in his most interesting and pathetic account, how his great-grandmother died of puerperal mania, leaving an only child, the grandfather of Thomsen. She had two sisters; both suffered late in life from mental aberration. His grandfather attained the age of 64, and had, by a healthy wife, 4 children, of whom Thomsen's mother was the eldest but one. The disease scarcely appeared in her or her elder brother, while it was strongly developed in her younger brother and sister: these two were weak in mind, but could fulfil the ordinary affairs of life. Thomsen's mother lived to the age of 72. She had 13 children, of whom 7 were attacked with the disease; of the others, a sister was slightly affected in mind, the rest were healthy. Of the children of Dr. Thomsen and his brothers and sisters, numbering 36, only 6 were affected. He had met with no cases, outside his own family, during a practice of 36 years. In his own case he has noticed that when the body is warm through exercise the contractions are less marked; they are worst when the temperature is low, or the body cold; in commencing chill; in the period of incubation or the prodromal stage of acute fevers; and after muscular exhaustion.

As to the primary site of the evil, we are reduced to statements of opinions. Thomsen suggests it is to be sought either in the spinal system or perhaps in the brain itself. He feels as if the impulse which arises from the will is "not transmitted by the usual path in the ordinary way to the organ which it is desired to set into activity;" or he would seek for "an original disease in the sphere of activity in the brain, the Will." Seeligmüller, though no change has been met with, ascribes the seat of lesion to the lateral columns of the spinal cord; to which opinion Erb inclines, while admitting it can only be decided by further observation.

The reflexes usually are normal. It is to be remembered that in some cases there is lordosis of the lumbar vertebræ; that in Peters' case the reflexes were much diminished; that, of Benedikt's cases, one suffered from vertigo and headache, and his second case was abnormally sensitive to pain; while in Vizioli's second case, the patient "was subject to sudden vertigo; had pain over the lower lumbar and upper sacral vertebræ, radiating to the iliac bones, without arriving at the nates, worse after walking, but not increased by pressure or by moving the limbs." No improvement has been recorded in any case.

Petrina on Sensory Disturbances from Cortical Lesions.

(*Zeitschrift für Heilkunde*, Dec. 1881. Rep. in *Neurol. Centralblatt*, 1882, No. 1.)—The author reports six cases of cortical lesions, in which there were sensory symptoms.

1. Softening limited to the superficial layer in the insula, the lower part of the anterior central convolution, and the upper anterior part of the inferior frontal on the left side. No other

lesions.—Paralysis of the right middle facial branch, aphasia. Anæsthesia of the right upper extremity, right face and nape of neck (faradic test), analgesia, loss of sense of temperature.

2. Necrotic patch, size of a pea, at the inferior third of right Rolando's fissure, the two central convolutions in the neighbourhood superficially softened. Left hemiplegia and hemianæsthesia.

3. Superficial circumscribed (embolic) softening of inferior left frontal convolution.—Aphasia, middle facial paralysis. Anæsthesia, as in Case 1, in right arm and upper half of trunk.

4. Small circumscribed patch of softening in cortex of left anterior central convolution, the first three convolutions of the insula, and the anterior upper part of the superior temporal convolution.—Right hemiplegia and hemianæsthesia; aphasia; ataxy of right arm after movement had returned.

5. Small hæmorrhagic tubercle, the size of a lentil, in the right superior parietal lobule. Hemiplegia and hemianæsthesia of left side. Hyperæsthesia to pressure and passive movements of left leg.

6. Caseous tubercle, the size of a hemp-seed, in Broca's convolution, surrounded by small hæmorrhagic spots. Aphasia, spasms of right arm and corner of mouth, followed by paresis. Anæsthesia of right trunk to pricking and temperature; hyperæsthesia of right arm to pressure and passive movements.

The author on the other hand adduces two cases of extensive lesions, the one in the anterior, the other in the posterior, cerebral convolutions, in which there was no sensory disturbance. Anæsthesiæ due to lesions of the posterior third of the internal capsule are characterised by their wider distribution than those just described.

[We have here a good instance of the kind of evidence and logical methods by which the theory of cerebral localisations is assailed. The common fault of the arguments brought to bear by its opponents is that they prove too much.—Rep.]

Remak on the Localisation of Lead Paralysis in the Lower Extremities. (*Neurologisches Centralb.* 7, 1882.) It had hitherto been considered that the tibialis anticus enjoyed the same immunity in saturnine palsy of the leg as the supinator longus does in the arm. The author himself had collected several instances to prove this. He finds, however, the rule to be not absolute, and describes a well-marked case of lead poisoning in which the tibiales antici were completely paralysed and electrically unexcitable, the extensors and peronei remaining intact. The flexors were likewise healthy, but the gastrocnemii were affected. This type of localisation is one frequently met with in atrophic spinal paralysis of children and of adults—a fact which affords another proof of the pathological relationship between these various conditions, and strengthens the author's view of the poliomyelitic nature of saturnine palsy.

Schulze on Tetany and the Mechanical Excitability of Human Nerves. (*Deutsche Medic. Wochenschr.* 20, 21, 1882.)—Prof. Schulze has been able to confirm on seven cases of tetany the discovery by Chvostek (cf. *Weiss, Sammlung Klin. Vorträge v. Volkmann*, No. 189) of the mechanical excitability of peripheral nerves (facial, nerves of extremities). He describes two of these cases in the present paper, and shows that the contractions obtained by percussing several nerves were not of a reflex or direct muscular nature. In the first the hyper-excitability had lasted a whole year. In the second a curious peculiarity existed, viz. the possibility of calling forth the spasmodic attack by compressing the carotid (the pupil of the same side was thereby dilated).

With reference to the mechanical excitability of the nerves, the author shows that it must be considered only as an exaggeration of a property possessed by the normal human nerve. In the healthy subject, percussion at points near the insertion of the zygomaticus major in the malar bone and near the triangularis menti usually bring about contractions. The nerve to the frontal muscle may occasionally be so excited. The axillary nerve in the supraclavicular fossa, the musculo-spiral over the humerus, and the ulnar at the elbow, may likewise be stimulated by percussion. Strange to say, the peroneus does not react to mechanical stimuli; the spinal accessory more readily so.

The "hyper-excitabilité neuro-musculaire" of Charcot differs from the normal or exaggerated mechanical excitability of nerves, inasmuch as it occurs only during the hypnotic condition, that it is manifested by permanent pressure (which has no effect in tetany), and that it is characterised by contrac*tures*, and not by physiological contrac*tions*.

A remarkable fact is that the mechanical hyper-excitability occurs sometimes in bulbar paralysis, and never in facial spasm and other hyperkineses. In tetany it is accompanied by an excessive reaction to electrical stimuli; the sensory nerves are never implicated.

The excessive excitability of nerves explains readily how attacks are brought on by active muscular exercise. The influence of arterial compression is less readily explained; experiments tend to show that mere anæmia of a part does not always cause the attack. Weiss's theory, that the irritation of the sympathetic may account for the result, is untenable.

Prof. Schulze argues to the peripheral nature of the disease from the following facts: occurrence of local paræsthesiæ, knee-jerk normal (in one case absent), absence of reflex hyper-excitability, frequent localisation in upper extremities, &c. We know nothing of the morbid changes occurring in the nerves in tetany. The treatment must be chiefly hygienic and of long duration.

Demange on the Relation of Senile Trembling to Paralysis Agitans. (*Revue de Médecine*, 58, 1882.)—The object of the author is to show that senile tremor is a true disease, which cannot be satisfactorily distinguished from shaking palsy. He proposes the term "Rhythmical oscillating tremor" as including both. He gives tracings obtained on the revolving cylinder of the various forms of tremor. Voluntary effort increases the amplitude of the waves in senile tremor, but does not always diminish them in paralysis agitans. In the latter disease the tremor may invade the muscles of the head, as shown in several cases observed by the author. [Luys, in a recent paper (*Encéphale*, 1881. 4), also considers senile tremor not as a physiological expression of decay, but as the manifestation of a pathological state allied to shaking palsy.—REP.]

Demange on the Loss of Teeth and Crises (Gastric and Laryngeal) in Locomotor Ataxy. (*Revue de Médecine*, March 1882.)

CASE 1.—Male, age 33. For eighteen years has had lancinating pains; for ten years ataxy. Anæsthesia and analgesia of the face, conjunctivæ, mucous membranes of nose and mouth. He has lost nearly all his *upper* teeth, which dropped one by one, every two months, in the space of about four years. He has had attacks of severe gastric crises and nervous cough. *Post-mortem.*—Posterior sclerosis of the cord extending into the floor of the fourth ventricle. The greater part of the nuclei are involved. Sclerosis of numerous bundles of fibres in the root of the trigeminus.

CASE 2.—Male, age 64. Syphilis thirty years ago. Twelve years ago "perforating ulcer" of foot, followed by lancinating pains and ataxy. Generalised anæsthesia, more marked in legs, fingers, and left face. All the teeth of the left upper jaw dropped a month ago. Fracture of neck of femur on falling from the bed. Four months after admission, death from asthenia. *Post-mortem.*—General atrophy of cord and posterior roots. Posterior columns sclerosed up to fourth dorsal vertebra—higher, Goll's columns only. Sclerosis extends to floor of fourth ventricle, with atrophy of the cells. Trigeminus and Gasserian ganglion on left side highly atrophic. Fatty degeneration of gastrocnemii.

Berger on Idiopathic Cramp of the Tongue and of the Cremasters. (*Neurologisches Centralblatt*, 3, 1882.)—The author remarks that spasms localised in the tongue alone are so rarely met with that their existence has been doubted. He describes two cases, however, where this condition was well marked. In both there was bilateral clonic spasm of the protrusors of the tongue. Reflex cremasteric spasms are not rare. But in two cases he observed the spasm was clearly idiopathic. It occurred both by day and by night, each attack lasting two or three minutes; the pain was very great. In one case atropia injections were successful; but failed in the other, where galvanisation (from the spine to the scrotum) afforded complete relief.

Vierordt on the "Shoulder-arm Type" of Paralysis. (*Neurol. Centralblatt*, 13, 1882.)—Erb has shown that in many subjects there is a motor point above the clavicle, opposite the transverse process of the sixth cervical vertebra (corresponding anatomically with the cord formed by the fifth and sixth cervical roots), at which faradisation brings about contraction of the infraspinatus, deltoid, biceps, brachialis internus and supinator longus chiefly, as well as sensations over the shoulder, and parts innervated by the median. On the other hand, a number of cases of atrophic paralysis have been described in which these very muscles were implicated. Hence it was concluded that in such cases either the grey matter giving origin to the motor roots, or the motor roots themselves of the fifth and sixth cervical nerves were the seat of the lesion. Vierordt describes a case which lends considerable support to this localisation of spinal influence. A man after an explosion was found to have received certain injuries. Among other symptoms was considerable swelling above the left clavicle, with loss of power in the corresponding arm. Four months afterwards he was found on examination to have several muscles atrophied and paralysed, viz. the deltoid, infraspinatus, teres minor, subscapularis, biceps, brachialis, supinators. They all gave degenerative reactions to electrical stimuli. Sensation was abolished over the deltoid; much diminished about the clavicle; diminished over the median area of the hand. Faradisation at Erb's motor point on the right side produced contractions of the muscles corresponding to those paralysed on the left side.

Bernhardt on a Case of Isolated Paralysis of the Supinator Longus. (*Centralblatt für Nervenheilkunde*, 15, 1882.)—A man had been stabbed from behind, the knife penetrating at the level of the fourth or fifth cervical transverse process on the left side. Among the other early symptoms was a severe pain in the left forearm, in the region of the supinator longus. Five months afterwards this muscle was found to be completely paralysed and atrophied, with degenerative reactions. All the other muscles had escaped, or had only been transitorily affected. Faradisation at Erb's supraclavicular point showed normal reactions in the usual set of muscles except the supinator. The deltoid, however, did not respond with normal readiness.

Schulze on the Morbid Anatomy of Infantile Paralysis. (*Neurol. Centralblatt*, 19, 1882.)—Prof. Schulze had the opportunity of examining the cord of a child who died three years after an attack of acute poliomyelitis. The case was a typical one, as testified by Prof. Erb and Dr. Hörman, who saw it from the outset. The atrophied muscles were chiefly the flexors of the foot and tibialis anticus on the right, the extensors of the foot on the left. The flexors of the thighs were also affected, as well as the triceps on both sides (slightly). The knee-jerk was present. The anterior horns of the lumbar region were much contracted; the lateral columns small and grey. Microscopically, the grey matter was found altered as far back as the substance of Rolando. The cells much atrophied; in places completely absent. The walls of the blood-vessels (chiefly those penetrating by the anterior fissure) were thick; here and there, masses of blood pigment. Anterior and lateral columns show diminution of nerve-fibres and increase of connective tissue, especially in the neighbourhood of the anterior horn.

Prof. Schulze insists upon the incompatibility of these changes with the view which makes infantile paralysis a parenchymatous, systematised lesion of the anterior horn. The inflammatory process does not start from the cells of fibres of the cornua, but in the district of the anterior vessels. Acute poliomyelitis is a kind of acute myelitis, characterised by its limitation to the anterior and lateral regions of the cord. The changes in the white matter are of a different nature as those of secondary degeneration, as well as regressive and differently distributed.

Waller on the Latent Time of the Opening Contraction.

(*Archives de Physiologie*, 1882. No. 3.)—The author has found on the human nerve that the latent time of the opening contraction was much longer than that of the closure contraction, and was led to investigate whether this indicates a persistence of the anelectrotonic state after the opening of the circuit. This persistence he demonstrated by the following experiment. A galvanic and a faradic (200 interruptions per sec.) current are placed in circuit, so that the exploring electrode contains the galvanic anode and faradic kathode. Their respective strength is so regulated that the anelectrotonic influence of the galvanic suspends the tetanising effect of the faradic. The galvanic current is then removed from the circuit by derivation, and the contraction following the physiological release of the faradic stimulus recorded on the cylinder. The author's tracings show that the anelectrotonic condition may persist ·05 sec. after the opening of the galvanic circuit. The latent time of the opening contraction exceeds that of the closure contraction by ·04 sec.

A. DE WATTEVILLE.

Marcacci, Experiments on the Cerebral Motor Centres.

(*Comptes Rendus de la Société de Biologie*, 1882. Nos. 4 *et* 5. Preliminary notice.)—Of the movements caused by electrical currents applied to the surface of the brain, the author sought to distinguish what are due to excitation of the cortex and what to that of subjacent parts, medulla and cord, and arrives at the conclusion that the effects of excitation are not altered when the cortex is rendered physiologically inert. The author's chief results are as follows:—The movements excited by electrical currents at the surface of the brain are similar before and after the cortex has been frozen by the spray until it is as hard as wood; a similar in difference is observed in anæsthesia, provided this be not so deep as to involve the suppression of spinal reflexes, and in paraplegia produced by freezing in a ring round the exposed medulla; ligature of the brachio-cephalic and left carotid does not alter the effect of currents applied to the brain surface, but if chloral is injected, which can now only act on the cord, cortical excitation becomes inefficacious. From these results the author concludes that the cortical substance is physiologically indifferent as regards surface excitation, which acts only on deeper parts, viz., on the medulla and cord.

Lewaschew on the Innervation of the Cutaneous Vessels. (*Pflüger's Archiv*, xxviii. p. 389.)—This memoir gives a detailed account of a laborious series of experiments carried on by the author during the last three years, for the purpose of defining the distribution of vasomotor nerves to the lower extremity. It appears from these experiments, that as a general rule the vessels of the various cutaneous districts are chiefly, if not exclusively, controlled by the nerves, which give to these districts their supply of sensory and other fibres. The sciatic has most influence on the cutaneous temperature of the toes and foot, less on that of the leg; the anterior crural has most influence on the internal surface of the thigh, less on the internal surface of the leg; the external cutaneous influences most the external surface of the thigh, the small sciatic chiefly its posterior surface. But while it thus appears that the vasomotor control of the limb is shared in by its various nerves, it also appears that the sciatic has by far the largest influence; that of the other nerves, including the ant. crural is relatively small, and not at all constant experimentally; whereas the experimental oscillations by section and excitation of the sciatic may have a range of as much as 15°, those effected by the anterior crural rarely reach 2°. Among other particularities the author records the following. It sometimes happens that within a given district certain points vary in an opposite direction to that of the general variation of temperature in that region; apparently this is due to the fact that the same paralysing cause, or the same stimulus, may act in either direction according to the peripheral state—constricting previously dilated vessels, dilating previously constricted vessels. After previous section and consequent degeneration of several nerves, the effect of excitation of a remaining nerve is not increased, i.e. this nerve acquires no compensatory increase of function.

Setschenow on Galvanic Phenomena in the Medulla Oblongata of the Frog. (*Pflüger's Archiv*, xxvii. p. 524.)—The cerebral hemispheres of a frog having been removed, and its spinal cord carefully exposed and isolated, the medulla oblongata is "led off" by unpolarisable electrodes applied to its section and to its surface. The primary deviation of the galvanometer reveals a current therein directed from intact to cut surface (i.e. from cut to intact surface within the medulla). Compensation having been established, the galvanometer shows that the original current is gradually decreasing, and the magnet is observed to oscillate somewhat irregularly with gradually increasing frequency. The oscillations finally take the form of true negative variations, which Setschenow considers to be dependent on spontaneous discharges of the nerve-centre. He supports this view by arguments and facts which appear fairly certain and conclusive. He proceeds further to justify the following statements. The extent of the negative variation stands in no proportion to the primitive deflection, but is closely related with the functional excitability of the medulla. The spontaneous nerve-discharges which underlie the variations occur only in the upper half of the medulla oblongata; the spinal cord, led off in a similar manner, does not manifest any such spontaneous variations. The apparently spontaneous effects are in reality effects of summating stimuli; discharges can be provoked by the vibrations of prolonged musical notes.

To study the effects of reflex excitation of the medulla, stimuli were applied to the sciatic nerve or to the spinal cord. Such a study is however attended with difficulties and uncertainties, which leave a good deal of play to the subjective factor in observation and deduction. Excitation of the sciatic may apparently provoke discharge or may interfere with spontaneous discharges; its effect upon them is thus somewhat similar to the effect upon the heart's beat of vagus-excitation.

According to the author, his observations prove, inter alia, the possibility of provoking electrotonus of nerve-centres.

A. WALLER.

BRAIN.

JULY, 1883.

Original Articles.

A CONTRIBUTION TO THE STUDY OF SYRINGOMYELIA (HYDROMYELIA).

BY PROFESSOR C. WESTPHAL.

It is well known that a dilatation of the central canal is not infrequently found in the fœtal spinal cord, especially in connection with other developmental irregularities of the cord. Its appearance in the adult is less common, and when found, it is often by accident, no symptoms having occurred during life to call attention to the cord. Not in all cases, however, in which the central canal of the adult cord has appeared enlarged, has the condition been one of simple dilatation (internal hydromyelia), as in the fœtus; most cases present, on the contrary, peculiar complications, which render it doubtful whether the cavities found represent congenital dilatation of the canal, or whether they are to be attributed to pathological changes which have occurred in later life.

The chief circumstances complicating the simple cavity-formation are a thickening of the tissues surrounding the cavity, and the appearance of new growths. There may also be grey degeneration (sclerosis) of the posterior columns, atrophy of the grey substance, hæmorrhages in or about the cavity, and sometimes the remains of acute or chronic circumscribed myelitis in the cord or medulla oblongata. Such pathological appearances can be attributed partly to the direct mechanical pressure of the distended cavity, and partly to inflammatory changes resulting from this pressure. In order to arrive at the right interpretation of the cavities and their mode of origin, those cases only have to be considered in which the formation of the cavity is complicated with the growth of a neoplasm, or with a thickening of the tissues around the cavity.

Hallopeau[1] had already in 1870 attempted to explain the formation of cavities in certain cases, otherwise than by a congenital dilatation of the central canal. The thickening of the surrounding tissues (sclérose diffuse periépendymaire) he assumed to indicate an inflammatory process occurring in later life; and thought that the new growth resulting from the inflammation could partly thicken and partly soften. The central canal, according to him, remains in parts unaltered, in others becomes dilated and destroyed, on account of the softening of the surrounding tissue, and of the pressure of the serum poured into its lumen; whilst the newly-formed connective tissue persists and increases. The central canal might thus, he thought, become irregular, extending backwards and partly losing its epithelium; while, on the other hand, its lumen might be encroached upon by the increasing tissue of the ependyma.

The question was revived by the studies of Simon[2] (who at the same time critically reviewed the earlier literature), and by my own investigations.[3] Simon, as well as myself, was able to distinguish a normal central canal in its usual position and *in front of* the cavity, from which we drew the conclusion that the latter could not represent a dilatation of the former. We found also in each case a new growth, as well as a cavity, in the spinal cord, with a probable connection between the cavity and the new growth, and we both endeavoured to show that the cavity was merely the result of a softening of the tumour beginning in its central part. This view was strengthened by the fact that, in certain sections, parts of the new growth were still recognisable on the inner wall of the cavity. One can see plainly in the illustration accompanying my former article (Plate III.) that there exists a large cavity in the upper part of the cord, surrounded by a thick capsule; lower down portions of new growth appear on its inner wall; and still lower, a consistent tumour, in which only a small canal is visible. In front of the cavity and tumour appears the normal central canal in its usual position. What supposition could in this case be more probable than that the original pathological condition was a new growth, which was gradually disintegrated from above downwards, and so led to the formation of a cavity?

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[1] "Contribution à l'étude de la sclérose diffuse périépendymaire;" 'Gaz. Méd.' 1870, Nos. 30, 32, 34, 35. "Etude sur les myélites chroniques diffuses;" 'Arch. génér. de Méd.' 1871, 1872.

[2] Th. Simon, "Beiträge zur Pathologie und pathol. Anatomie des Central-Nervensystems;" 'Arch. für Psych. und Nervenkrankheiten,' V. i. 1874, p. 109.

[3] Westphal, "Ueber einen Fall von Höhlen und Geschwulstbildung im Rückenmark," &c.; ibid. p. 80.

It is nevertheless remarkable that in most recorded cases the seat of the lesion is constant, being generally found in the *posterior columns* of the cord; further, that, apart from the central canal, cavities are found in the new growth which show the remains of a lining of *cylindrical epithelium*. Although Simon was inclined to draw the conclusion that the cavity formed by the softening and absorption of a new growth could under certain circumstances become lined with cylindrical epithelium—a view not supported by other facts—the thought suggests itself, on the other hand, that we have to do either with congenital anomalies in the spinal cord, such as a double or triple central canal, or with a constriction of the canal by the pushing in of a new growth. That a divided central canal may occur in a spinal cord presenting no further anomaly, is a fact sufficiently evident, however we may explain its existence, so that the supposition of Simon to explain the occasional lining of the cavities in cases of syringomyelia with cylindrical epithelium, is unnecessary.[1]

Finally, embryology has taught us how it is that the cavity almost unexceptionally has its seat in the posterior columns; for at a certain stage of development the central canal extends as a fissure between the posterior columns, to the periphery of the cord, and becomes enclosed at a later period by the transverse development of the posterior columns. If this development is interrupted and the fissure is only partly obliterated, there will remain a cavity or tube in the posterior columns.[1] This view is in most perfect accordance with the result of Leyden's[2] examination of two cases of congenital internal hydromyelia. There could be no doubt that the condition in both cases was one of true hydromyelia, for both children (aged respectively two and a half and two years) suffered from congenital hydrencephalocele, which eventually caused death; and the cephalic tumour consisted of a hydrocephalic dilatation of the cerebellum, which, as is well known, leads, by implication of the fourth ventricle, to internal hydromyelia.

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[1] Compare, amongst others, Pick, "Ueber die Entstehung eines mehrfachen Central-Kanal;" 'Arch. für Psych. und Nervenkrankheiten,' VIII. 2, 1878, p. 285. Pick describes here a triple central canal. I have myself recently seen a similar case. Compare 'Charité Annalen,' 1883.

A review of the alterations found in the spinal cord in these two cases of real hydromyelia shows a peculiar analogy with those found in the syringomyelia of adults. With regard to this relation, Leyden lays stress on (1) the resemblance which a cavity extending along the cord from the lower extremity of the fourth ventricle downwards, and leaving the lumbar region unaffected, bears to the condition found in syringomyelia; (2) the position of the cavity in the posterior columns; (3) the fact that (as is also generally the case in syringomyelia), in the two cases mentioned, the canal was surrounded by a layer, more or less thick, of consistent tissue, which had in one case a horny and in the other a gelatinous character resembling that of the ground-substance of the spinal cord. This tissue developed itself around the canal, and in certain sections took on the character of a tumour. In certain portions of this mass appeared[3] (as in the previously mentioned cases of Simon and myself) a breaking down in the centre, so that a cavity appeared within the new growth, situated entirely in the posterior columns; (4) the layer which surrounded the canal appeared in the one case thin and tough, in the other soft but greatly developed, a difference found also in the cases of syringomyelia; (5) the cylindrical epithelium lining the cavity (although there can be no doubt that it arose from the central canal) had in places disappeared, from which it follows that one can draw no conclusion from the absence of such epithelium in determining the nature of a cavity; (6) Leyden lays stress also on the relation between the affection of the posterior columns often found in syringomyelia (especially of the columns of Goll), and the condition of the same parts in cases of developmental irregularity; he considers the non-union of these columns in the median line as the starting-point of the affection.

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[1] Compare Waldeyer, "Ueber die Entwicklungsgeschichte des Central-Kanals im Rückenmarke." Virchow, 'Arch.' 68, p. 20, 1876.

[2] Leyden, "Ueber Hydromyelus und Syringomyelie;" Virchow, 'Arch.' 68, p. 1, 1876.

[3] At least Leyden explains the fissure in this way.

Finally, Kahler and Pick have shown in a number of cases that considerable dilatation of the central canal, resulting simply from development and causing no pathological changes, may exist unobserved in the adult; also that, notwithstanding the existence of a normal central canal, a central cavity may have arisen from the enlargement of the central canal, and that, even in the case of cavities occurring in a situation unusual for the enlarged central canal (for instance in a posterior horn), one cannot deny the possibility that we have to do with diverticuli from the central canal.

After all this, there can hardly be a doubt that the view formerly adopted by Simon and myself,[1] and which seemed supported by the facts observed, must be given up, in that the syringomyelia can no longer be regarded as quite independent of changes in the central canal, but a certain relation to such changes must be acknowledged.

The following description of two cases will serve as a contribution to the solution of the question under discussion.

Case I.—Hohman, aged 67, a carpenter, came to the Charité, June 6, 1871; was treated in the department for nervous diseases from Jan. 10, 1872, till Feb. 1, 1873; from this time, till May 1874, was in one of the general wards; after which he was treated in the department for nervous diseases until his death, which took place May 24, 1874.

From the history taken in the general ward in 1871, it appears that the patient had suffered from intermittent fever, which he contracted at Wilhelmshafen a year before he came to the hospital, and of which he had had an attack three weeks before admission. He also experienced difficulty in breathing, and complained of a sensation as if inspiration were hindered beneath the manubrium sterni, this sensation following a persistent cough. He was described as a decrepid individual. Objectively, stridor, superficial breathing, enlargement of the spleen, and soon a great increase in the amount of urine, which had a low specific gravity (1006–1015). The maximal amount in 24 hours was 5400 cc., the minimal 2500 cc. Patient had an insatiable appetite. He did not know how long the polyuria had existed.

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[1] Cf. Fr. Schulze, Virchow's 'Archiv,' vol. 87, p. 510.

On June 30, 1871, it was noticed that the legs were swollen; patient felt as if he were encircled by an iron band. Examination showed no abnormality of sensation or localisation. The temperature, as far as noted, never rose above 37·2°, and averaged 36·5°.

Neither laryngoscopic nor other examination revealed the cause of the stridor. Substernal struma was suspected, but objectively only bronchial catarrh, with tough viscid sputum, could be established.

In August codein was administered, after which the appetite lessened, and the quantity of urine fell to 1600–2800. The appetite did not increase after the codein was discontinued (August 17). The urine gradually became more abundant, so that, for example, the quantity on Sept. 15th was 4900.

No sign of paralysis of lower extremities before Sept. 23rd, 1871. Two days before this date he had fever, which set in with a temperature of 39·8°, and on the evening of the 23rd reached 40·0°. On this day were noted pains in the lower extremities and inability to stand. On the next day the temperature was normal. No cause for the fever was found. After this, recurring headaches were noted, which lasted several days. The appetite had returned on the first of October. The polyuria remained unaltered.

On Nov. 4th, 1871, a careful examination was made, in which appear the following statements:—Marked tremor in lower extremities, and severe headache, the latter of which "rises from the small of the back." The patient cannot walk. In bed he can lift the legs with a zigzag movement to an angle of 30°. Pains in the legs. Sensibility in the lower extremities seems absent, but examination shows that deep pricks of the pin are felt. The results are somewhat uncertain, on account of the inattention and lack of intelligence of the patient.

The lower extremities lie always extended, and it is nearly impossible to bend the knee; the movement being attended with great pain. Flexion at the hip-joint is possible, but difficult, and painful. The ankles are also stiff. Rotation is the only voluntary movement possible in the lower extremities. Patient says he is unable to stand. Even when placed on the edge of the bed the knees are not flexed, though one can with an effort bend the leg to a right angle; left to itself, it returns to the extended position. If one raises the whole limb in bed, and lets it go suddenly, it does not drop at once, but gradually (and sometimes by successive jerks), as if let down by the will of the patient. The cause is probably a stiffness in the muscles of the hip-joint. Patient is unable to stand; says that when he could still walk in the general ward, he noticed trembling in the limbs. On micturating, patient complains of feelings of pressure and constriction.

The quantity of urine on Jan. 11th, was 4900 cc.; Jan. 12th, 5600 cc. (specific gravity 1006); Jan. 13th, 5500 (specific gravity, 1000). On the 12th and 13th we found by Trommer's test a substance reducing cupric oxide; the polarisation apparatus showed 1·3 per cent. of sugar.

On the 16th, 6600 grammes of urine were passed, with a specific gravity of 1007, no precipitate. On the 18th, 6000 grammes (specific gravity 1007); a distinct precipitate by Trommer's test.

The notes upon the case after return to the general ward are confined to measurements of the urine, which were very variable, ranging from 1200 cc. to 5500 cc. The treatment consisted principally in injections of morphia, in the course of which an erysipelas was developed, with high fever. This complication was treated by injections of carbolic acid.

On the 7th of May, 1874, the patient was sent back to the clinic for nervous disease. Increased quantity of urine seems not to have been noticed recently, although occasional enuresis rendered exact measurement of the urine impossible. Abnormal constituents no longer found in urine. The following notes were taken in the Clinic for nervous diseases on May 21, 1874.

Temperature.—Morning, 37·0°; evening, 37·2°.

Patient says he cannot lift his legs. Slight flexion only possible in knee-joint; somewhat more in ankle and toe-joints. On attempting to perform dorsal flexion of the foot, one experiences a resistance from the tendo Achillis which prevents the complete flexion. This resistance is still more marked on the right. No trembling in lower extremities. Limbs emaciated, as is the whole body. Faradic excitability of the muscles normal. Pricks of the pin in the lower extremities cause a grimace; other tests of sensibility impossible. The function of the upper extremities is affected; he can perform all movements, but with very little strength. No disturbance in articulation from affection of the tongue, and no atrophy of the same; no partial atrophy of skeletal muscles noticeable. Urine very dark, brown-red, without sediment, 1030; no albumen, no biliary colouring matter, no sugar.

Patient complains since yesterday of difficulty in deglutition, and eats only fluid food. Experiences a sensation as if he were "laced in" in the region of the manubrium sterni.

May 22, *Temperature.*—Morning, 36·5; evening, 36·3. The twenty-four hours' urine cannot be measured, because patient has passed the greater quantity in bed; there seems, however, to be no increase. If one raises the thigh, the leg drops by its own weight, and no stiffness appears in the knee-joint. Patient called attention to a swelling in the submaxillary region; and examination showed enlargement of the lymph glands. Mercury ointment and a Priessnitz compress ordered. The glandular swelling the next day decidedly increased, and filled the space between the ascending ramus of the jaw, and the cervical vertebræ, extending downwards to the angle of the jaw.

Marked collapse; hands cyanotic; radial pulse hardly perceptible, 78, sometimes intermitting. Patient died on the morning of the 24th.

Autopsy.—Exceedingly emaciated subject, nowhere œdema.

On both thighs swellings the size of walnuts, which on section prove to be hæmorrhages into the subcutaneous cellular tissue between skin and fascia. In the region of the angle of the lower jaw a swelling, shown by section to be a purulent infiltration of the enlarged parotid gland. Lower down, separate swollen glands. Swelling of follicles at the base of the tongue. Cartilages of the larynx partly calcified, mucous membrane of larynx normal. A stenosis in the upper part of the trachea, caused by projection of the cricoid and three first tracheal cartilages, narrowing the lumen of the trachea. On the outside of this prominence lies the lower end of the left lobe of the thyroid cartilage. Thyroid cartilage not hypertrophied, on section normal. In the œsophagus, somewhat below the tracheal stenosis, a pouch the size of a hazel-nut, with a neck of narrower diameter. *Lungs,* marked emphysema anteriorly, and chronic catarrh. Central softening of the right suprarenal capsule. *Kidneys,* hyperæmic. Mucous membrane of *stomach* thickened and reddened. Nutmeg *liver.* *Spleen* friable. *Bladder* contracted, containing a moderate quantity of urine; mucous membrane normal. Intestines and genitals not examined. Subpericardial fat tissue strongly developed. *Heart* large, flaccid, right ventricle greatly dilated; papillary muscles somewhat flattened. Heart walls rather thick, heart substance pale. Valves normal.

Spinal cord.—On opening the dura mater a small quantity of serous fluid escapes. The dura is everywhere smooth, nowhere adherent. The pia is not thickened in any part. The cord itself is in the upper part noticeably soft and glistening. On the surface of the cervical enlargement, the portion of the right posterior column limiting the median fissure shows a dirty-brown colour on a width of about two millimeters. This discoloration extends to about 29 cm. above the conus medullaris. Sections in the upper part of the cervical enlargement show an exceedingly glistening surface, above which projects a tumour of dirty-brown colour, measuring 4 mm. transversely, and 2–3 mm. longitudinally. The tumour lies in the region of the right posterior column, the surface of which remains free. It involves the grey substance, but appears not to reach the central canal. The same brown mass appears on all sections made below this point, extending in some places beyond the median line. A section 4 cm. above the conus medullaris presents microscopically no abnormal appearance. The consistence of the cord is here good, while 14 cm. above the point of the conus medullaris, the cord is already softened, and shows the circumscribed red mass already described. Somewhat below this point in the lumbar enlargement both posterior columns present a brownish discoloration. The anterior columns are everywhere free, as are also the anterior horns. Nothing abnormal in the left lateral column; the right appears somewhat glistening and compressed, discoloured in spots. Nothing abnormal upon the roots or the cauda equina.

Cranium.—Dolichocephalus, somewhat asymmetrical, the right parietal bone being rather more prominent than the left. The diplöe on both sides of the sagittal suture, about 2 cm. posteriorly to the coronal suture, has almost disappeared. The sutures are externally well preserved, on the right strongly reddened. The frontal and both parietal bones are covered with red points and small prominences; on the right parietal bone appears one large and one small exostosis.

Brain.—The dura mater is strongly adherent at limited points in the frontal region. Pacchionian bodies along the longitudinal suture, corresponding to the thinning of the skull. The external surface of the dura mater shows nothing else abnormal. Fresh coagula in the longitudinal sinus. Inner surface of dura mater smooth, no pseudo-membraneous deposit.

Pia mater shows nowhere opacity nor thickening; its vessels are moderately filled; can be peeled off without loss of substance. Neither the vessels nor the nerves at the base of the brain present any abnormal appearance. The convolutions are rather small and pale; the grey substance of medium thickness; no infarctions in either the grey or white substance of the hemispheres. The white substance is moderately rich in blood. Nothing abnormal in the large ganglia. The right posterior column of the upper cervical cord shows the same discoloration as is seen in the cervical enlargement. One section appears, as above described, a dirty-yellow consistent mass projecting from the cut surface in the region of the right posterior horn, and the inner part of the right posterior column. The columns of Goll in the medulla oblongata appear discoloured. At the apex of the calamus scriptorius the sections present no abnormal appearance, nor does the floor of the fourth ventricle; sections through the pons and peduncles normal.

Fresh microscopical examination of the apparent tumour in the upper part of the cervical region shows everywhere numerous brown-red pigment masses, which lie imbedded partly in coarsely fibrillated connective tissue. In places the ground-substance is not fibrous, but finely granular and streaked, resembling neuroglia. The pigment masses which, with a magnifying power of 250 diameters, appear round and remarkably similar in size, are partly arranged in rows, as if escaping from vessels. In a few places it is evident that they lie in or upon the walls of the vessels. In the great majority of the masses this arrangement does not appear, but they lie irregularly and in heaps on the ground-substance described. In the preparations made by teazing out with needles, the vessels do not appear notably increased.

Examination after the hardening of the cord shows a new-growth, extending from the upper part of the cervical to the lumbar enlargement, and occupying the posterior part of the cord. The lowest section on which it is visible lies 11 cm. above the apex of the conus medullaris, in the right posterior column. The situation of the new-growth varies greatly, and on some sections two separate masses appear; the variations in seat and form of the tumour are so great as to make it hardly possible, or at least very tiresome, to describe each separate section offering peculiarities, those which have been pictured sufficing to give a general idea of these variations.

One sees that the new-growth (Fig. 1, upper part of cervical enlargement) lies principally in the right posterior column, and here extends to the grey substance, but it encroaches also upon the left posterior column. Its posterior boundary is pointed, and extends, less markedly developed, to the periphery of the region of the column of Goll. In Fig. 2 the growth occupies equally the anterior parts of both posterior columns, has displaced the grey substance of both horns, and extends, pointed, backward to the periphery. In Fig. 3 the posterior columns are included to a still greater extent, especially the right, and the commissure as well as the grey substance on the right is lost in the new growth. In Fig. 4 (about 27 cm. above the apex of the conus medullaris) two distinct masses are visible: one, of rounded form occupying the anterior part of both posterior columns; the other, situated on the inner boundary of the right posterior horn, long and narrow: in a section made somewhat deeper, the new-growth is situated no longer at the boundary of the posterior horn, but within the same, having taken its place. In Fig. 5 the mass in the right grey substance unites with that in the posterior column (here again principally in the right), thus causing a destruction of the grey substance except an outer strip; apart from this is seen a small mass of the growth in the left posterior horn. On deeper sections through the dorsal region, the growth gradually leaves the grey substance, leaving it at last quite free; and in the lumbar region there, separates itself again, a mass which lies at first in the anterior part of the posterior columns; it runs, pointed, backwards, ending at the periphery of the cord, and comes to lie more and more in the right posterior columns (Fig. 6), and at last, growing smaller, but preserving its form, it lies only in the middle and posterior part of the posterior columns, while the point no longer reaches the periphery.

Irregular as are its form and seat at its different heights, the tumour lies nowhere in the anterior regions of the cord, and particularly nowhere in the anterior or lateral columns; even in the sections in which it involves the anterior grey substance, the new-growth appears to have pushed forwards from behind.

Already with the naked eye one can distinguish an outer mass, coloured strongly red by carmine, and forming in places a broad and in places a narrow ring; and a delicately coloured transparent inner part. The latter appears to contain a split, partly simple and partly branching (the same is seen in the rounded mass in Fig. 4). This apparent split shows in many sections, even to the naked eye, peculiar zigzag lines. No real split, however, exists, and the appearance of one is only the result of the imperfect colouring in the inner parts, and in some sections apparently the result of partial separation, due to mechanical influence. It is only in Fig. 6 that the new-growth appears to consist of a regular mass; and even here one sees with a higher power an irregular part, very narrow, and easily distinguishable by its transparency and delicate colouring. This part is broader several sections above Fig. 6, and more plainly marked.

The central canal is everywhere, excepting where the new-growth encroaches deeply upon the commissure, plainly visible as a central thread of ependyma without special growth in its neighbourhood. Wherever the new-growth reaches the anterior commissure, and involves the portions behind the commissure, the canal fails, and no sign of it is visible in the growth. The structure of the growth is very peculiar, complicated, and difficult to analyse. The bundles of connective tissue and the vessels form truly labyrinthic convolutions. My friend Professor von Recklinghausen (of Strassburg), to whom I had an opportunity of showing sections, had the kindness to communicate at my desire the result of his examination. This communication I here insert.

"The difficulty of explaining the sections is very great; the relations in the four sections given me are so various, that it is hard to bring them under one head. I have attempted again and again the explanation from the beginning, hoping to reach the true solution. The following is now my opinion:

"In all sections through the tumour appears a fibrous substance more or less rich in pigment, very poor in cells, with here and there rather larger cells, which represent perhaps ganglion cells altered in shape. This tissue stands in close connection with the neuroglia of the surrounding posterior columns, in which the atrophied nerve-fibres are recognisable, so that the tissue makes altogether the impression of being formed by the neuroglia, and resembles in texture an inflammatory product of the spinal cord. The peculiarity of the formation consists in two things: 1st, the serpentine canals, lying close together and partly in connection with each other; probably branches of blood-vessels distinguished by an extensive, very firm, and almost structureless layer of varying thickness, which follows the almost unaltered intima;

practically no trace of elastic tissue or unstriped muscular fibres appearing, so that it is impossible to decide whether we have to do with veins or arteries, probably, however, the latter; the lumen is very narrow, contains no distinct blood-corpuscles, and is perhaps at least in part obliterated. 2nd, the peculiar papilla-like, or better, shirtfrill-like bodies, which appear in two places in the sections. The body appearing in the section marked (*x*) forms a completely closed ring, whose centre is quite filled with delicately-fibred connective tissue. The ring of the second body is on one side not closed; here, rather, the delicately-fibred connective tissue filling the centre is continuous with the connective tissue imbedding the whole body. Also here throughout, no lumen, no fissure. Each ring is composed of feebly-striped, transparent tissue, apparently wanting in cells. This tissue is not stained by carmine, and resembles the tissue already described in the walls of the vessels, differing only in that its appearance is more glistening. I cannot but give it the name *sclerotic connective tissue,* although it lacks the firmness and strong glance of sclerotic connective tissue in other organs (skin, serous membranes). This sclerotic tissue makes in the most complicated form *folds,* not properly papillæ; on the tops of the folds, and often in the valleys between the folds, the superficial layer is delicately striated, so that here the impression is given of vertical section through layers of flat epithelium, but neither defined cells nor elastic tissue can be recognised. I recall similar forms, especially as regards the folds, only in the walls of canals; for instance, on the inner layer of arteries, whose entire walls have been by some process thickened. The same appearance may be artificially produced on cross-section, through vessels by folds in their walls. At first, having these thickened vessels in mind, the thought suggested itself to me that perhaps these appearances represented a very large vessel, with thickened walls and lumen filled with adhesive connective tissue. Still, the diameter of the closed ring is so great as hardly to be attained even by a dilated blood-vessel in the spinal cord. There remained then only the supposition that another sort of canal gave rise to this appearance: it was probably a syringomyelia, as supposed by yourself, whose lumen had originally at least a diameter equal to the longest diameter of the present ring. Further, one must suppose that the inner layer of the wall surrounding the cavity took on a sclerotic character, while the outer wall, or, what is the same thing, the imbedding, almost adventitious connective tissue layer increased immensely in volume, so that the inner layer became folded, and the lumen narrowed. Finally then, the lumen must have disappeared through the formation of adhesive tissue, which afterwards in some places increased (also with breaking through of the sclerotic layer from outwards inwards, or in the opposite direction), and became continuous with the external basement substance. The situation of the pathological formation, together with the extent of the affection, seems to me to warrant the assumption that the whole formation represents a syringomyelia, with consecutive chronic inflammation thickening the walls and obliterating the lumen (periependymic sclerosis). The great difficulty lies in the unusual layer of sclerotic tissue in the wall (sclerotic, of course, in the common meaning of the word among pathological anatomists); still I remember to have seen preparations with Leyden, I think they were from his Case 3, 'Klinik der Rückenmarkskrankheiten," II. 454, in which such sclerotic tissue existed. I used at that time the term, but do not remember with certainty whether the tissue only existed on the vessels—it is certainly not rare for it to be found on them in other inflamed organs."

To assist in understanding the descriptions given of the microscopic appearances, I add a *figure* (7) which presents a portion of the new-growth lying near the right posterior horn (from Fig. 4, Hartnack, IV. oc. 2). One sees the papilla-like formation springing inwards from the strongly coloured tissue. Surrounding this formation, and pushing into its depressions, appears a layer-like tissue, not stained by carmine, but retaining the chromic-acid colouring. The central portion between the two irregular edges is filled with a finely striped, feebly-coloured tissue.

I add some details to this description, which fully coincides with the results of my own examination. On the sections in which the apex of the new-growth extends to the periphery of the posterior columns (Figs. 1–3), large separate groups of nerve-tubes are surrounded by bundles of thick fibrillar connective tissue. The bundles of nerve-tubes, on the other hand, are penetrated by stripes of fibrillar tissue, which limit smaller groups of nerve-tubes in the bundles. In certain spots the groups of nerve-tubes thus limited present a normal appearance, the medullary sheath being plainly coloured by the potassic chromate; in other places the cross-section of the tube is decidedly lessened, and instead of the yellow medullary sheath, one sees the contents of the tube not coloured at all, or feebly red; in some sections no axis-cylinder can be made out in these small tubes; in other sections the axis-cylinders are plainly visible as little red points, much smaller than the normal axis-cylinders in the unaltered nerve-tubes of the posterior columns (Cf. Fig. 8). Over and between the separated nerve-tubes lie numberless cross-sections of blood-vessels, with adventitia enormously thickened and fibrillated. The bundles of nerve-tubes in Figs. 1 and 2, appear as small holes (*a*), the vessels as small red points Fig. 2 (*b*). The posterior columns themselves, wherever they are free from the growth, are unaltered. No grey degeneration appears in the columns downwards from Fig. 1. Above the commencement of the new-growth in the upper cervical region, on the other hand, the posterior columns appeared at the autopsy grey and transparent; these portions were, however, not available for microscopical examination. The grey substance of the anterior horn is anteriorly everywhere intact, except in those sections in which this substance is encroached upon by the new-growth. It is especially to be noticed that the ganglion cells, even with a low power, appear in normal number and form, and with plainly marked off-shoots. In the posterior part of the anterior horn, on the other hand, the grey substance, at the points of junction with the new-growth, is, by the loss of medullated nerve-fibres, more strongly coloured, of a spongy appearance, and presents a thick network of the finest fibres and spider cells, at the border of which tissue appear single ganglion cells, somewhat smaller, flatter, and containing less protoplasm than normal. The alteration in the grey substance is, however, everywhere limited to the immediate neighbourhood of the tumour.

Case II.—Stimmel, a carpenter, aged 58 years, admitted into the Clinic for nervous diseases of the Charité, August 28, 1875.

As the history of this case has already been published by my former assistant, Dr. Wernicke,[1] I shall not repeat it here, especially as the symptoms observed during life were not due to the lesion in the spinal cord, but to a tumour in the pons, and the publication was limited to the results of this tumour, while the case interests us here especially on account of the cavity in the *spinal cord.* Only the description of the tumour in the pons is therefore quoted here.

Autopsy: Brain.—From the floor of the fourth ventricle to the left of the median line projects a tumour-like elevation, 1½ cm. long and 2 cm. broad. This elevation appears to be formed by the junction of three smaller ones. Its posterior boundary lies nearly 1 cm. distant from the apex of the calamus scriptorius, its anterior boundary is 1 cm. distant from the corpora quadrigemina. The surface of the tumour is permeated with blood-vessels, varying in size and moderately filled with blood. The tumour projects nearly ½ cm. into the fourth ventricle, is quite firm to the touch, and grey-red in colour. No striæ acusticæ visible on either side. The tumour does not extend into the crus cerebelli ad pontem, but the left corpus restiforme is in its inner portion evidently filled with new growth. No sign of new growth found on section of the separated pedunculus cerebelli. A section through the middle of the tumour shows that it does not reach forwards as far as the anterior division of the pons; to the right it hardly passes the median line, and to the left it is limited by the lateral boundary of the pons. Section through the region of the crossing of the pyramidal tracts shows no evidence of new growth. No further pathological changes found in the brain beyond a moderate dilatation of the ventricles.

The dura mater of the *spinal cord* presents nothing peculiar; the veins on the posterior surface of the pia mater in the lower part of the cord are somewhat tortuous; the pia itself is transparent, not thickened, nowhere adherent to the dura. Sections through the cervical region just below the crossing of the pyramidal tracts show a grey discoloration of the portions of the posterior columns bounding the posterior septum. On sections through the cervical enlargement appears a soft, grey-red mass, occupying the whole anterior part of the posterior columns, which mass reaches laterally to the posterior horns, pressing them somewhat outwards, and anteriorly extends without defined boundary into the commissure. The posterior horns are still plainly recognisable, also the anterior horns, although parts of the latter seem involved in the new growth. The posterior portions of the posterior columns appear free from new growth, but a brown discoloration extends along the septum. The tumour described is plainly visible in the lower cervical, but ceases at the commencement of the dorsal region, sections below being those of an apparently normal cord. The consistence of the spinal cord in the regions occupied by the tumour is soft, in form flat, and elsewhere its form and consistence are normal. The posterior roots present no abnormal appearance.

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[1] 'Arch. f. Psychiatr. u Nervenkrankh,' vol. vii. p. 513.

Microscopical Examination.—No sign of new-growth appears in the cervical region; above the cervical enlargement, on the other hand, the middle portions of the posterior columns show grey degeneration, as is seen in the illustration, and the anterior boundary of the degenerated portion being at the commissure. In the cervical enlargement (Fig. B) appears a cavity situated in the posterior columns, surrounded by a ring of varying breadth, coloured by carmine. This ring is lined by a narrow zone of tissue which has not taken the staining. The remaining portions of the columns of Goll are degenerated. The seat of the cavity in the following sections, as well as the breadth and form of the ring surrounding it, may be seen in Figures C, D, E; one sees that in Figure E the cavity comes to lie in the right grey substance, and a new mass appears in the right posterior columns. This mass is of the nature of a new growth, and shows a delicate colouring at its edges. The cavity ended in the dorsal region; its termination could not, however, be followed, as the part of the cord in which it ended was not available for examination. The central canal (with cylindrical epithelium) in Fig. A, appears somewhat widened, and in certain sections in this region, is filled partly with red blood-corpuscles, and partly with a homogeneous, clear red-coloured mass, as if coagulated, and containing single blood-corpuscles. Elsewhere the central canal lies in its normal position as a central ependymal thread, excepting in the sections where the circle of new-growth (as in Fig. E) extend far into the commissure so as to destroy the integrity of the latter. The ring of new-growth consists of an entanglement of fibrillar connective tissue, in which numberless nuclei are seen after staining with hæmatoxylin. On some preparations the ring appears in spots to be formed from distinct layers in the process of preparation. The outer part of the ring (in which the large number of cross-sections of vessels is noticeable) runs into the surrounding tissue in a zigzag manner, or with a network. The adventitia of the vessels is everywhere remarkably thickened. Here and there at the periphery of the ring, and in the grey substance in its neighbourhood, can be seen masses of dark-brown pigment, and minute fresh hæmorrhages (collections of red blood-corpuscles). In Figs. A and B, the inner border of the ring consists of a narrow strip of connective tissue with parallel fibres, coloured feebly or not at all by carmine, but well by hæmatoxylin, which brings out also numberless nuclei; on other sections no such inner boundary can be demonstrated. Of special interest is the fact that on several sections—for example, that represented in Fig. B—appear two bodies, not very far apart, directly under (outside) the inner rim which is not coloured by the carmine. These bodies resemble closely the body near the right posterior horn in Fig. 4 (Case I.), the microscopical appearance of which is shown in Fig. 7. There appears in them the same shirtfrill-like folds, with the same corresponding layer-like tissue as in the former place, only that the central finely-striped, delicately red-coloured tissue fails. The boundary line of the bodies towards the lining membrane, under which it laid, was partly simple, and in part appeared studded with similar papilla-like projections and depressions, similar to those in the inner side of the body itself, surrounded in like manner by the layer *a*.

As we have seen, the first case is one of *new-growth*, the second one of *cavity-formation*. If we notice, however, the situation of the new-growth in the first case, its form and its extension through the spinal cord, the idea involuntarily presents itself, that the case of new-growth is allied to that of cavity-formation. Its seat in the posterior columns and posterior horns correspond exactly to that of the cavity described; the form of the new-growth has also, especially in the cervical enlargement, a noticeable resemblance to the form of the cavity which one finds in this region in cases of syringomyelia. The fact also that the growth in the one case, as the cavity in the other, ends in the lumbar region, shows a correspondence with most cases of syringomyelia. To this is added the peculiar formation of the tumour, which in many sections shows inside a particular body. All these facts make it highly probable that this body originated in a cavity, which originally has been a diverticulum of the central canal, as we have become acquainted with above, and that the tumour became gradually filled with the "sclerotic" tissue. The surrounding ring of connective tissue, strongly coloured by carmine, represents the ring which one commonly finds around a syringomyelia. We can only surmise the exact mode of occurrence of these changes (compare the remarks of Recklinghausen above).

The relationship of the new-growth in the one case, with the cavity in the other, is finally shown by the discovery of bodies, as above described, in the surrounding ring of the second case, closely resembling those described in the first case connected with the tough (sclerotic) tissue forming the inner border of the ring.[1] One can imagine that here the filling up of the cavity was interrupted, and that the shirtfrill-like folds had only become developed in separate spots, while in general the inner sclerotic rim laid smoothly on the outer ring.

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[1] This article was already written, when Dr. Pick showed me some preparations of Case III. in his work mentioned above (Kahler and Pick, 'Beiträge für Pathologie und patholog. Anatomie,' &c., Leipzig, 1879, p. 113), in which were present completely analogous, indented formations, in this case, however, starting from the inner border of a ring bounding a central cavity. They protruded into the lumen of this cavity; in some places there were remains of epithelium on and between the indentations. Cf. the interesting paper of Fr. Schulze quoted above, and a case related by Strümpell, 'Archiv für Psychiatrie und Nervenkrankheiten,' vol. x., p. 704.

There can hardly be any doubt that the changes in the spinal cord were congenital, and one must therefore assume that disturbance of development of the central canal ensued with cavity formation, accompanied by a tendency to development of fibrous new-growth. This development is often considerable in the surroundings of the cavity, and characterised by great vascular development and pigmentary deposit. The inner layers of the wall of new-growth take on meanwhile the peculiar sclerotic character described. In other cases there seems to be a tendency to the development of another form of new-growth (glioma, myxoma). Hæmorrhages often appear to accompany these processes.

The fact that in the second case a tumour was found in the pons, as well as a cavity in the spinal cord, might perhaps tempt one to conclude that a tendency to tumour-formation existed at the same time in different parts of the central nervous system. Were, however, the opinion correct which was formed from the histological examination; that in this case we had to do with a solitary tubercle of the pons, the idea of such a tendency must be given up. To-day the question could be definitely settled by the presence or absence of bacteria.

No symptoms appeared in the first case which could be attributed to the spinal-cord affection; the syringomyelia then ran its course here, as already often observed, quite without symptoms. The patient in the first case came to the hospital on account of his difficulty in respiration, and the discovery of the symptoms of sensation and motion was rather accidental, considering the mental condition of the patient. To sum up these disturbances, we find, paresis of the lower extremities, with appearance of stiffness; weakness of upper extremities, and a diminution of sensibility, whose boundaries could not be accurately determined on account of the patient's mental condition. There was no musclular atrophy, properly so-called (degenerative atrophy); the wasting of the subcutaneous tissue and muscles was general.

A symptom of especial interest was the increased quantity of urine and the occasional occurrence of sugar, which one might well be tempted to connect with the alterations in the spinal cord; for, according to the observations of H. Dickinson,[1] dilatation of the central canal in the dorsal and lumbar region, together with proliferation of the lining epithelium, are noticeable, though not constant, in the post-mortem appearances of diabetes mellitus.

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[1] Compare Senator, "Diabetes Mellitus und Insipidus;" Ziemssen's 'Handbuch,' xiii. 2, s. 189.

EXPLANATION OF THE ILLUSTRATIONS.

Fig. 1–6.—Cross-sections through the spinal cord of the first case (cervical and dorsal regions). Low power. (The details are as seen with Hartnack II.)

Fig. 7.—A part of the body (*c*) in Fig. 4, seen with Hartnack IV., ocular 2, tube drawn out.

Fig. 8.—Nerve-tubes surrounded by the fibrillary tissue of the new growth and deprived of their medullary sheaths, the axis-cylinder visible in many as a point. This view represents the point (*a*) in Fig. 1. (Hartnack VII.), ocular 3, tube drawn out.

Fig. A.–E.—Cross-sections through the spinal cord of the second case (cervical and dorsal regions.

Cc. The somewhat widened central canal.

a. Inner border of the ring.

b. Hæmorrhages.

[Translated by D. L. WALTON, M.D., *Boston*, U.S.A.]

Fig. 3.

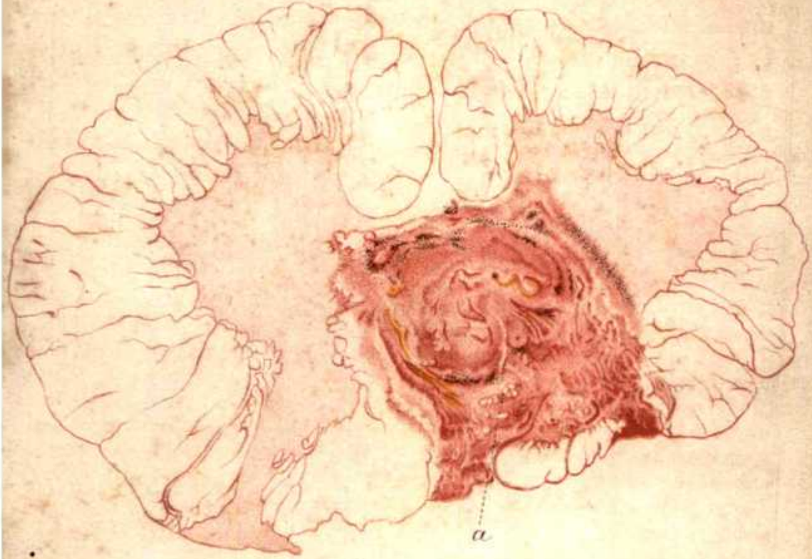
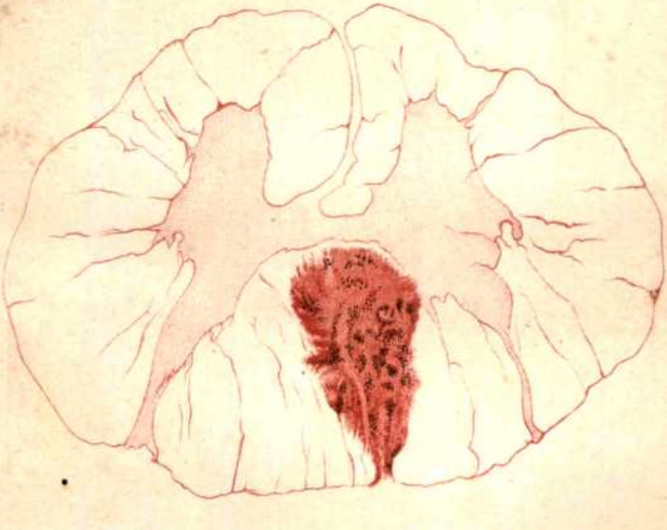


Fig. 6



C. Laue lith.

Fig. 2.

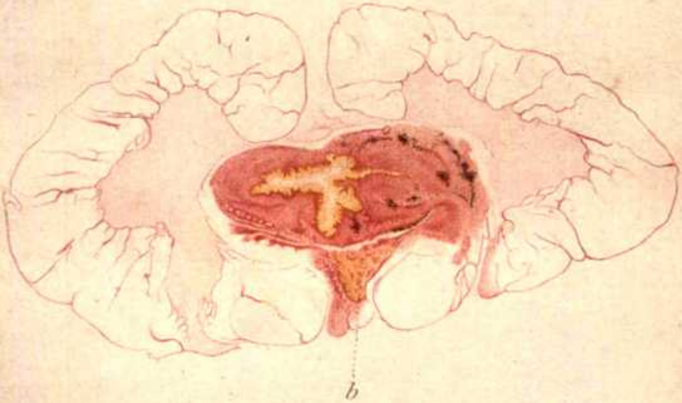
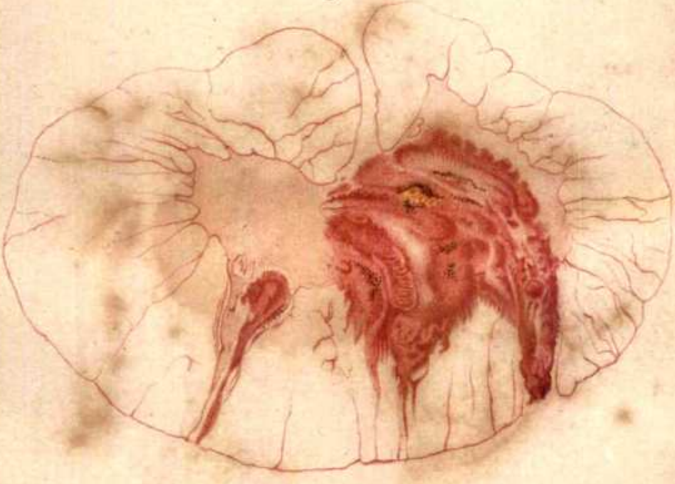


Fig. 5.



Westphal on Syringomyelia._ Brain, vol. VI.

Fig. 1.

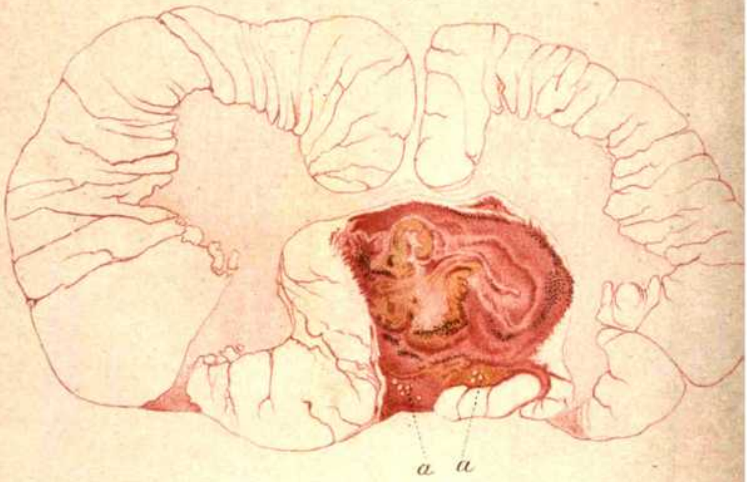
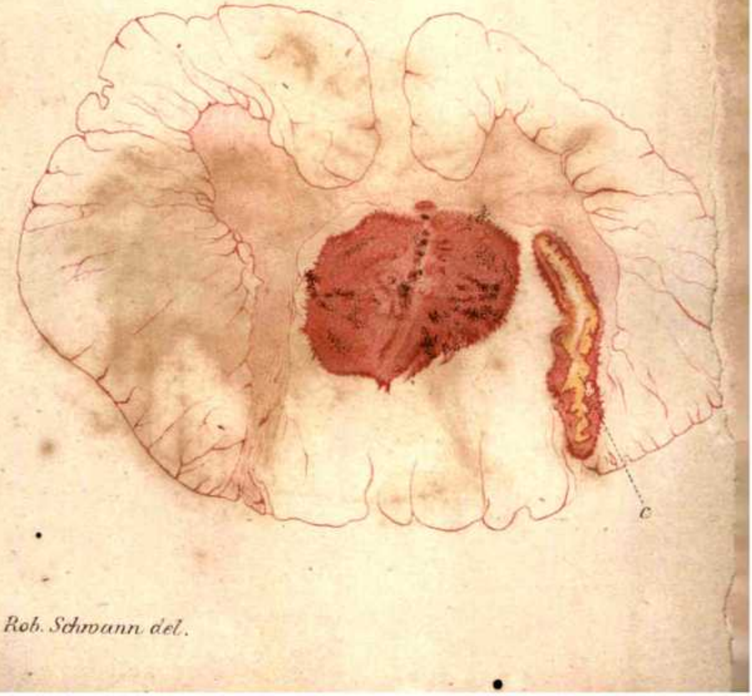


Fig. 4.



Rob. Schwann del.

Fig. A.

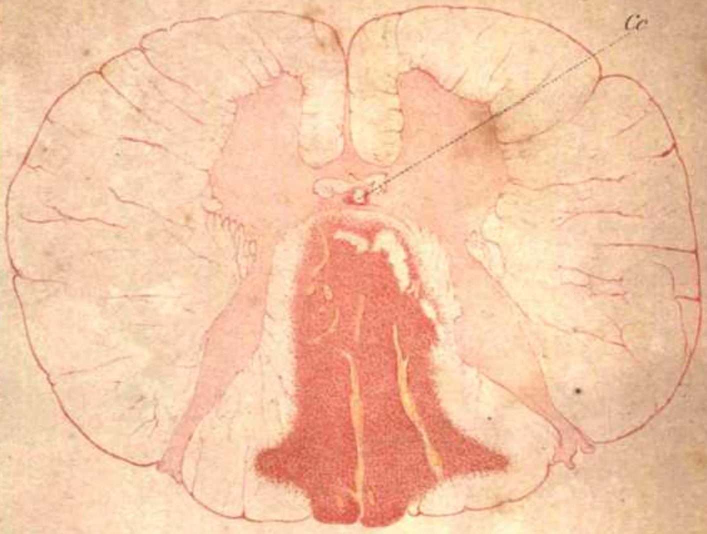
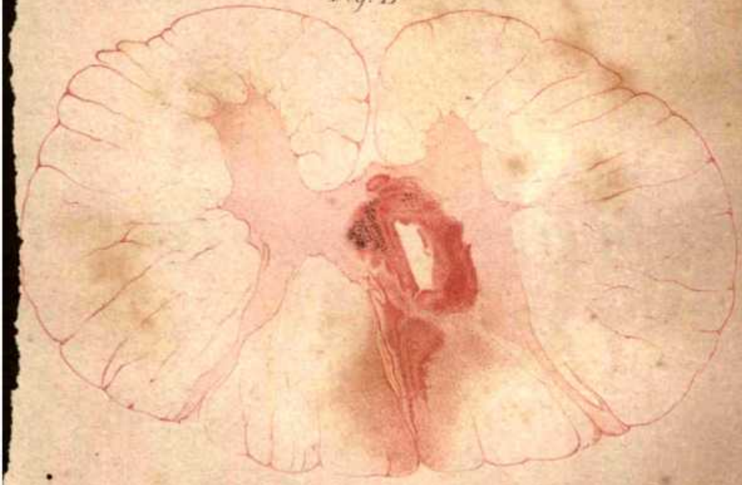


Fig. E



C. Laue lith.

Fig. B.

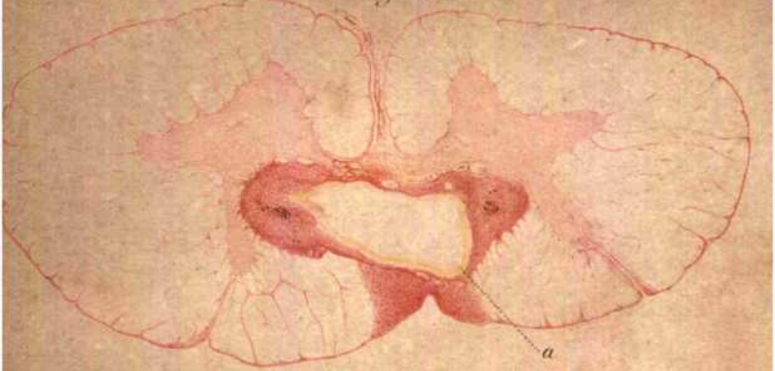


Fig. 8.

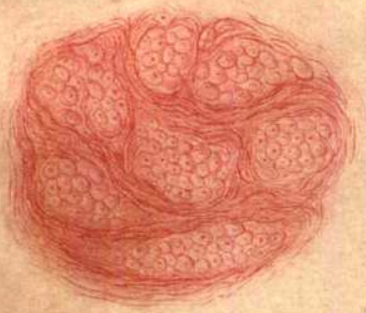
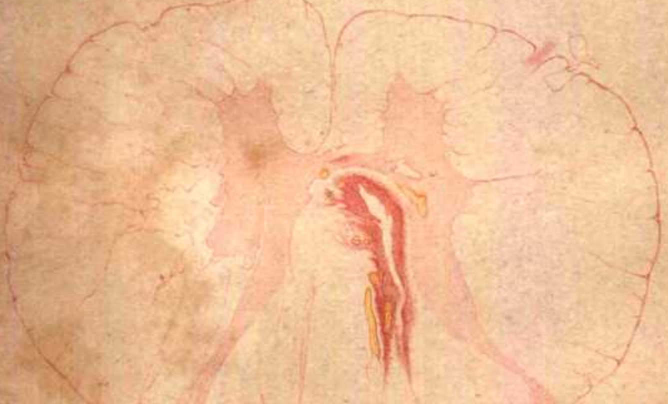


Fig. D.



Westphal on Syringomyelia._Brain, vol. VI.

Fig. 1.

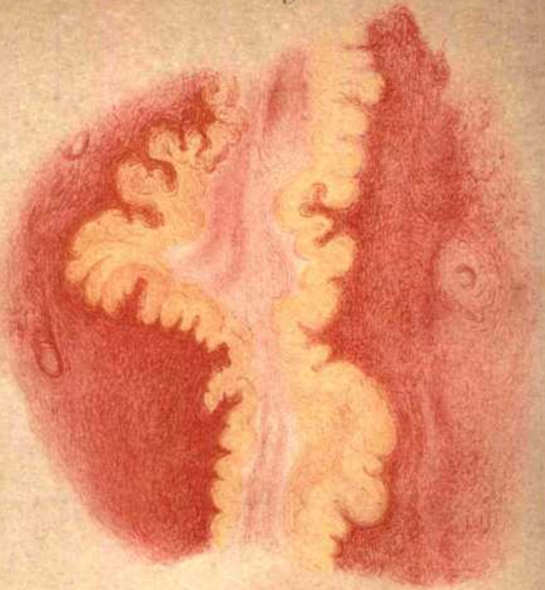
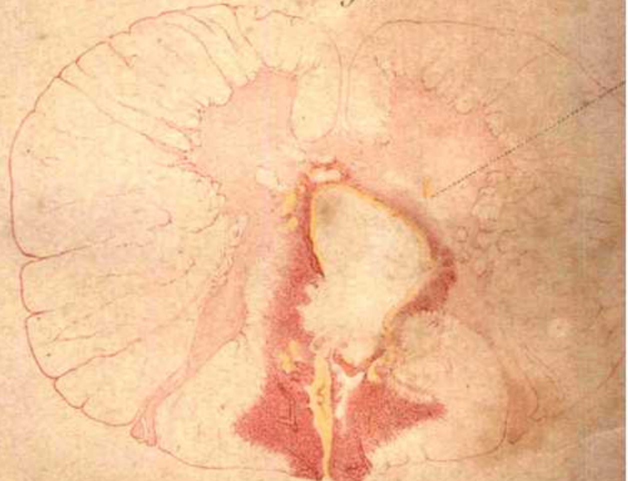


Fig. C.



Rob. Schwann del.

CLINICAL REMARKS ON TUMOURS INVOLVING THE PARTS IN THE NEIGHBOURHOOD OF THE THIRD AND FOURTH VENTRICLES AND THE AQUEDUCT OF SYLVIUS.

BY J. S. BRISTOWE, M.D., F.R.S.

THE cases which I am about to narrate and discuss were cases which interested me much during life; and are all the more instructive that they each became elucidated in the course of time by a post-mortem examination. The first case was one in which a tubercular mass had destroyed the corpora quadrigemina, and by pressure displaced the iter downwards and forwards; the second was one in which a tumour sprang apparently from the valve of Vieussens and processus e cerebello ad testes, and had invaded the corpora quadrigemina and cerebellum, and become adherent to the floor of the fourth ventricle; and the third was one in which a tubercular growth occupied the third ventricle and implicated the adjoining optic thalami. The fourth and fifth cases scarcely belong to the same category; inasmuch as the fourth was a case of tubercle of the cerebellum, and the fifth a case of chronic hydrocephalus. But I have ventured to add them to the others because, in the former the cerebellar tumour pressed upon the floor of the fourth ventricle, to which fact probably some of the symptoms attending it were due; and in the latter, owing to obliteration of the iter, the fourth ventricle formed an independent cyst, and thus its most interesting pathological feature was the presence of disease in the vicinity of the lesions presented by the other cases.

The first case was that of a little boy of seven, whose illness terminated fatally after six months' duration. The earliest symptom of his malady was, it was said, loss of power in the right arm and leg coming on suddenly. But before long all his limbs became paretic and tremulous on movement; and his head and neck also displayed tremors when unsupported. Further, he showed marked symptoms of ophthalmoplegia externa: protrusion of the eyeballs, ptosis, and paralysis probably of all the ocular muscles, though mainly of the superior and internal recti. Moreover, the eyes had a tendency to act independently of one another. Sight, however, remained perfect; the pupils (though at times unequal) acted to light and to accommodation; and there was no optic neuritis. His speech was drawling, and attended with tremors of the lips. There was absence of headache, vomiting and convulsions. Among subordinate symptoms, either present from the beginning, or coming on in the course of the case, were drowsiness, indistinct facial paralysis, exaggeration of the knee-jerk in both legs, with stiffness of the right, and loss of control over the evacuations. During the last few days of life acute symptoms came on, with considerable rise of temperature, which was due to the supervention of tubercular meningitis.

Now it was not difficult, during this patient's life, to make a shrewd guess as to the seat of his intra-cranial lesion, and even as to its nature. A young child is much more likely to suffer from a tumour than from sclerosis; and a tumour is, in such a case, much more likely to be tubercular than of any other character. That the child was suffering from a tubercular growth, was rendered almost certain later in his career, when symptoms of meningitis were added to his other symptoms. At any rate it was assumed when he first came under observation that, notwithstanding the absence of headache, vomiting, optic neuritis, and fits, he was suffering from cerebral tumour. The seat of the tumour was indicated by the ocular paralyses. It was evident that the nerve-trunks were not involved, and that the nerve-nuclei were not destroyed; and hence, either that the tumour was higher up or more anterior than the nuclei, or that it simply compressed them. The partial, and to some extent variable, facial palsy, with the absence of implication of any of the cerebral motor nerves below, and of any of the nerves of special sense, further seemed to indicate that the disease was in the corpora quadrigemina or their immediate neighbourhood. The presence of universal muscular weakness, with tremors, and the absence of actual paralysis of the limbs, indicated rather symmetrical compression and irritation of the motor tracts, than their actual destruction by a tumour.

At the post-mortem examination there was found, in addition to recent tubercular meningitis, a solitary tubercular mass, about as large as a marble, imbedded in the corpora quadrigemina, destroying these bodies, and displacing and indenting the tracts of nervous matter situated to the outer side of, and below, the aqueduct of Sylvius.

The second case was in many respects much more interesting than the first. It was that of a young lady of twenty, whose illness extended over rather more than two years; and whose condition, up to two months before her death, was attributed to aggravated hysteria. The first symptom noticed was loss of power in the middle finger of her left hand, which interfered with her playing on the piano. To this succeeded deafness of the left ear, and dimness of the left eye; which before long also presented an inward squint. Later, she complained of numbness in the left arm and leg; and of some stiffness, associated with tremors, in the legs. The phenomena above described became slowly developed, one after another, during a period of eighteen months. During the next four months, however, the progress of her disease was rapid. She became deaf and blind; the left eye turned outwards instead of in; she spoke in a drawling, whining tone, and was at times incoherent; and her limbs became generally very weak, and all her movements were attended with tremors. On admission, two months before her death, she was for the most part in the condition just described. She could stand and walk when supported; but all her limbs were weak, and these and her head and neck trembled, as in disseminated sclerosis; she was quite deaf and blind; there was no ptosis, but the eyes diverged and acted at times independently of one another. The paralysis was most complete in the right external rectus; but apparently all the muscles of the right eye were weak. There was probably also some weakness of some of the muscles of the left eye, which also presented rotatory nystagmus. The pupils were unequal; and there was double optic neuritis. She drawled in speech; and was affectionate, not to say amorous in her manner. There was slight paralysis of the right facial. When she had been in hospital about a fortnight, a mattery discharge took place from her right ear, which continued until her death. In this case, as in the last, the patient was very drowsy; and there was (at any rate to within a day or two of her death) no headache, sickness, or convulsions. During the the last few days of life, the weakness in her legs increased; she had violent general tremors at times; she presented difficulty of swallowing; the veins of the inner halves of the eyelids became swollen; and her temperature rose.

In this case, as in the former one, it seemed clear during life that the patient was suffering from a cerebral tumour. And the association of absolute deafness, with the ocular troubles, partial facial paralysis, and general paresis and trembling, rendered it pretty clear that the tumour was seated somewhere about the fourth ventricle, and was of considerable extent. There were some points in the case, however, that added a little obscurity to it. Was the loss of power, limited to a single finger (which was her earliest complaint), referable to the tumour, which, as it grew, ultimately caused her other symptoms? Could the persistent, though lately developed, otorrhœa have any relation to a tumour occupying the substance of the brain, or was there a mere chance association between them?

The post-mortem examination showed that the diagnosis made during life was accurate; and suggested an explanation of some of the other incidents of the case. A largish growth was found springing apparently from the valve of Vieussens, and the processus e cerebello ad testes, which was adherent to the floor of the fourth ventricle in nearly its whole extent, and had involved the testes and cerebellum. But it was found also that the dura mater at the base of the skull, in every one of the fossæ, was the seat, in a greater or less degree, of the same kind of disease. The discharge from the ear was doubtless dependent on the latter complication. I could not, however, determine that any of the paralytic phenomena were due to involvement of cranial nerves, or narrowing of their foramina of exit.

The third case was that of a little boy, four years old, and was obviously (as will be seen from a perusal of the history) one of tubercular growth in the brain. The symptoms of his cerebral affection lasted nine months. They began with tremors, and slight loss of power in the left arm and leg; which were followed in the course of a month or six weeks by tremors in the limbs of the opposite side. These tremors were like those of disseminated sclerosis. The left side of the face also presented spasmodic movements at this time. About six months from the beginning of his illness (the symptoms having made gradual progress during this time), the left arm and leg had become rigid; the tremors of the right arm and leg had increased; there was some twitching in the left side of the face, with paresis of the left facial nerve; he had commencing double optic neuritis; he drawled in his speech; he was extremely drowsy; and he passed his evacutions into the bed. These symptoms presented little change, up to a week before his death; when his temperature rose, he vomited, and presented other symptoms of acute meningitis, of which he died. In this case, as in the others, there were no fits, no headache, no sickness, and the appetite was extremely good. It is important also to note that there was no paralysis of any cerebral nerve. There was, no doubt, weakness of the left facial, but this was clearly hemiplegic.

Where was the tumour that caused the symptoms above described? I confess I thought it would be found in the cerebellum. And I still think that this diagnosis was justifiable. The tumour, however, was found occupying the third ventricle, and involving to some extent both optic thalami. And I am inclined to think that, with the guidance of such cases as the first two narrated, a correct diagnosis might have been arrived at.

In reviewing these cases collectively, it is impossible not to see that they presented many features in common, which were probably due to the fact that the same tract or tracts were involved, and the same tract or tracts avoided, in each; and, at the same time, many points of distinction which were clearly referâble to the additional involvement in each case of special centres or regions.

The chief points of resemblance between them were the following:—general paresis, with tremors like those of disseminated sclerosis; drawling speech, reminding one somewhat of that observed in myxœdema, and in one case attended with marked tremors of the lips, and thus presenting a close resemblance to the speech of general paralysis; remarkable drowsiness, with more or less emotional disturbance and loss of control over the emunctories; and in association therewith absence of anæsthesia, headache, convulsions and sickness, and the presence of an excellent, if not voracious, appetite.

The chief distinctive features, which may be attributed to difference of seat of lesion, were the following. In the first case, paralysis of both third nerves, and probably of both fourth nerves, without implication of the irides or muscles of accommodation; due probably to compression of the nuclei of origin of these nerves. In the second case, deafness on both sides, with some involvement of one, if not of both, third nerves; due probably to extension of disease into the auditory nuclei, and to pressure on the nuclei of the third nerves. In the third case, the absence of all specific phenomena; owing to the fact that the tumour was seated above the origins of the motor cranial nerves.

There were other phenomena presented by the cases, to which more or less importance may be attached; but to which, on account of their frequency in brain affections, I have not ventured to assign specific value. Among them were, paralysis of the sixth nerve, observed in one, if not in two, of the cases; incomplete facial palsy, probably independent of direct involvement of the seventh nerve; optic neuritis, which was marked in two of the cases; blindness, probably the consequence of neuritis; and the symptoms due to the supervention of meningitis observed shortly before death in the two tubercular cases.

The fourth case presented a close superficial resemblance to the third. The patient was four years old; his illness lasted for nine months; he had in the beginning, and for some months subsequently, tremors of both arms, and of the head and neck, which (so far as the arms were concerned) gave way to rigidity some time before death; his speech was drawling and monotonous; and he had double optic neuritis with blindness. Further, he never suffered from sickness. But he suffered constantly from what none of the other patients had experienced, namely headache and epileptiform seizures; he presented, more markedly than any of them, the so-called "cerebellar gait;" and, in the course of his illness, paralysis of the left sixth and seventh nerves came on. Pathologically the case was most like the second; for although the tumour (which was of somewhat larger size) was a cerebellar tumour, it exerted manifest pressure on the floor of the fourth ventricle, mainly its left half. It was doubtless owing to this pressure that the patient had paralysis of the left sixth and seventh nerves; and probably owing to the same cause that he suffered from tremors of the head and neck and arms, and that his speech was drawling.

The appearances discovered after death showed that, in the fifth case, the lesion from which the patient suffered was of old date. It consisted, in fact, of dropsical effusion into the lateral and third ventricles, into the fourth ventricle, and into the central canal of the spinal cord. An interesting point, however, in the case was that the dropsical accumulation in the fourth ventricle was cut off from the accumulations in the neighbouring cavities.

The symptoms (excepting that the head was unusually large) did not point to the nature of the patient's malady. He died of a convulsive attack, of which he had had several during the last four or five months of his life. And his other chief symptoms were, feebleness and unsteadiness of gait, rigidity of the neck, headache, occasional double vision, and slight feebleness of mind with tendency to be noisy at night. He had no paralysis and no tremors.

CASE I.—*Tubercular Tumour of the corpora quadrigemina, followed by tubercular meningitis.—Paralysis of both third nerves, with ptosis, and tremors of head and neck, arms and legs.—No fits.—Sickness and headache absent until the last.—No optic neuritis.—Death.—Autopsy.*

Edward B., a schoolboy aged 7, was admitted under my care on the 2nd of August, 1882.

His illness began about the latter end of May. He is said to have gone to bed well, and to have got up the next morning with inability to walk properly, and with marked weakness in the right arm and leg. About a week later his eyes became affected, and gradually assumed their present condition. His speech became impaired at the same time. He has had no headache, no vomiting, no fits; his memory has not suffered, and his temper has remained good; his appetite has been good, his bowels constipated. He has been drowsy latterly, however; and for the last few days has passed his evacuations into the bed. He has also been getting weak and thin; and tremulous movements of the limbs have been gradually developing.

The child is thin, and has a large head; which, when he sits up, is usually thrown somewhat backwards. He is drowsy and dull; but when fully roused appears to be perfectly sensible. The limbs are all weak; but there is no distinct paralysis of any of them. When he sits up, the head trembles; when he moves his arms, the movements are attended with coarse undulatory tremors; and when he stands or walks, similar tremors affect the legs, and become transmitted to the body generally. The eyes are both unduly prominent; there is ptosis on both sides; and also distinct paralysis of the superior and internal rectus of the right eye, which looks outwards, and apparently a less degree of the same paralysis on the left side. The right pupil is slightly larger than the left; but both pupils act to light and accommodation. Sight appears to be perfect. The mouth is drawn a little to the right when the child laughs, but there is no other evidence of facial paralysis. The tongue is protruded straight. Speech is slow, drawling, and monotonous. No difficulty in swallowing. Hearing unaffected. No impairment of sensation. Superficial and tendon reflexes normal. Tongue clean; appetite good; bowels confined. Urine 1030; lithates, no albumen. No sign of thoracic or abdominal disease.

For the next three weeks there was no marked change in his condition. The description above given of him was in the main confirmed. Drowsiness and apathy varied in degree, but were generally present; he had no headache, or sickness; his evacuations were sometimes restrained, but occasionally escaped unwittingly; the tremors continued, but it was noticed also that the lips trembled when he spoke; the paralytic condition of the eye-muscles remained much as it was; and it was ascertained that fundus of the eyes were healthy. It was noticed, further, that the occasionally sudden spasmodic tremors convulsed his whole frame. But there was no apparent loss of consciousness in these attacks. He retained his good-humour.

About the 25th of the month, and for a day or two afterwards, he complained of some occipital headache.

During the next month, again, there was very little change on the whole.—except, perhaps, that drowsiness and disinclination to speak or to take notice of things increased; that he became irritable, and apt to cry out at times, and even to try to bite when annoyed; that his evacuations were now all passed involuntarily; that he got less and less able to support himself in the sitting posture; and that his tendon reflexes became exaggerated. The tendency to retract the head continued; the tremors of his head and neck and limbs did not diminish, possibly increased; the slight paralysis of the right facial nerve was still observable; and the prominence of his eyeballs continued. On the 26th of September his eyes were examined by Mr. Nettleship, who reported as follows. "All the movements of the eyes are more or less imperfect, unless it be the movement downwards, which is better executed than the rest. The defect is greatest in the right eye, both as to the levator palpebræ, the superior rectus and the internal rectus. The paresis of the external recti is considerably less marked than that of the superior and internal recti. The right eye is directed outwards, and stands on a rather lower level than the left. The pupils are equal, of natural size, and act well to light. Action to accommodation I could not determine. The oculo-motor symptoms point to disease higher up than the trunks of the nerves, and missing the centres for the iris and ciliary muscles." Sight seemed to be perfect, and there was no optic neuritis.

For the rest of September, and during the whole of October, the condition of the child scarcely presented any change. There was no increase of paralysis; he still seemed to understand, but was unable or indisposed to reply, and rarely spoke. But his attention could be aroused, and he could be made to laugh. The eyes were examined on several occasions, and the fundus was always found normal. He never had any distinct indication of headache, and never any sickness, or any fits.

About the 10th of November, it was noticed that he was a good deal weaker; that he had ceased to know his mother when she came; but that nevertheless he could see, and if a piece of cake was held up before him would grab at it; that the right pupil was much larger the left, and that both acted badly to light, and that there were no ophthalmological changes; that the mouth twitched a little to the left; that the patellar tendon reflexes were brisk, and that there was a tendency to rigidity of the right leg.

On the 17th it was remarked, that the boy had been getting more and more drowsy and indisposed to speak, though now and then inclined to be noisy; that the paralytic condition of the eyes remained, and that the ptosis was becoming more marked, especially on right side; that the tremulous movements had largely diminished, and that he still had no sickness, no obvious pain in head, and no fits.

On the 18th he vomited; and again he vomited on the 20th. The vomiting still continued; on the 22nd it was observed that he often put his hands to his head as if in pain, and that the tremors in arms and head had returned. After this he became weaker, and quieter, refused his food and continued to vomit occasionally; and his temperature, which had nearly always been normal, rose. On the 25th it reached 100; on the 26th, 101·2; on the 27th, 101; on the 28th, 101·6; on the 29th, 102; and on the 30th (the day of his death) it rose to 103·8. He died comatose, and without convulsions. The condition of his retinæ was not ascertained subsequently to the 11th of the month.

Autopsy.—The convolutions of the brain were flattened; the surface was dry and slightly congested, and the veins were engorged. The membranes at the base were thickened, and lymph was present in the subarachnoid tissue about the interpeduncular space. There were a few miliary tubercles at the base of the brain; which were most abundant in the Sylvian fissures. In the right fissure, just by the anterior convolution of the island of Reil, the tubercles were aggregated into a small tumour. The corpora quadrigemina, and apparently these alone, were occupied by a globular cheesy mass, about as large as an ordinary marble. This lay between the upper surface of the bodies and the iter, and was invested above and below by a thin remnant of the normal textures. The position of the corpora quadrigemina, as seen from above, was occupied by a smooth rounded expansion, which was due to the presence of the tumour and the consequent effacement of the proper contours of the part. The lower part of the tumour was received into a cup-like depression of the structures below the level of the iter; the latter passage being obliterated by pressure and forming from before backwards the segment of the circumference of a circle, with the convexity facing downwards and forwards. The substance of the brain was generally healthy; but the lateral ventricles were distended to some extent with fluid.

With the exception that the pleuræ presented adhesions that the lungs were thinly studded with grey tubercles, and that numerous larger yellow tubercles were scattered through the spleen, the abdominal and thoracic viscera were healthy.

CASE II.—*Tumour of valve of Vieussens, involving cerebellum and corpora quadrigemina, and adherent to floor of fourth ventricle.—Similar growth in dura mater of base.—Blindness, optic neuritis, paralysis of ocular muscles, deafness, tremors of head and limbs, and loss of power in limbs.—Mental affection—Discharge from right ear.—Death.—Autopsy.*

Lily M., a girl aged 20, was admitted under my care on the 4th of April, 1882.

Two years ago the third finger of her left hand became weak (not flexed), so as to interfere seriously with pianoforte playing, of which she was very fond, and in which she was clever. Later, she complained of dimness of the left eye, and deafness of the left ear, with sounds in it as of bells ringing. These symptoms persisted, and at the end of a year she began to see double. Five months later (seven months ago) she presented a manifest inward squint of the left eye. About this time, too, she began to experience difficulty in walking; there was some tremor in the legs, they occasionally crossed one another when she attempted to walk, and she had to grasp at things for support; and she complained of numbness in the left arm and leg. She also suffered from fatigue, required assistance in dressing, and looked pale and ill.

From Nov. 20 to Feb. 8, she was away from home; and there is no record of her symptoms, beyond the fact that she gradually got worse. When she returned, the arms, and head and neck, were tremulous as well as the legs; and the left eye turned outwards. Her sight and hearing also were more defective than they had been; she was so weak on her legs that she could not walk without assistance; her speech was affected; and she talked at times incoherently.

From this time she rapidly got worse. Early in February the catamenia ceased; about the same time she complained that she was absolutely blind and deaf; and her talk was at times nonsensical and incoherent. She spoke in a drawling, whining tone; and talked constantly of herself, and in complaint of her blindness and deafness. She seemed not to recognise people, and was apt to give them new names, by which she continued to know them. She has generally recognised her mother, however. Her mother believes that she sees and hears, at any rate at times. Since January she has occasionally passed her evacuations in bed.

She is naturally of a gentle and affectionate disposition; and, excepting when (on one or two occasions) she has given way to temper, she has retained these qualities throughout her illness. She has never shown symptoms of hysteria, unless any of the above phenomena are to be regarded as hysterical; which was the view of those who sent her to the hospital.

The patient is a fair, well-nourished girl. She can walk with assistance in a shuffling way; but for the most part keeps her bed. She is blind and deaf, and speaks in a slow drawling manner. She has marked tremors of the arms (like those of disseminated sclerosis) when she uses them, and of the head and neck when she sits or stands. The legs also tremble to some extent. There is no loss of feeling; indeed perhaps a little hyperæsthesia. The pupils are unequal, the left being larger than the right, and do not react to light. The eyes diverge; this appears to be due to an external squint of the right eye, the movements of which seem generally restricted. But it is difficult to be sure which ocular muscles are affected, inasmuch as the patient cannot see, and cannot hear so as to do what she is told. There is slight rotatory nystagmus of the left eye. Double optic neuritis is present: much swelling extending some distance from the disc, very tortuous and dilated veins, and several striated hæmorrhages. There is slight facial paralysis on the right side. She is constantly talking in a slow whining fashion, mostly on matters that concern herself, and often asks questions and to be kissed. If not gratified, she turns crossly on her right side. She does not try to do anything for herself; but if food be placed in her hand, she then puts it into her mouth. The legs and arms are all weak, but there is no definite paralysis. The patellar[1] and plantar reflexes are absent, and there is no ankle-clonus.

No affection of thoracic or abdominal viscera. Tongue clean. Appetite fair. Bowels confined. Urine pale, acid, no albumen. Temperature 99·2.

During the next fortnight her symptoms accorded in the main with the above description. She has occasionally got up; but generally lay in bed on her right side. She was very drowsy, and at times difficult to rouse. When awake, she was sometimes noisy, and called out for her mother. She seemed to know that she was left among strangers, but evidently did not know where she was. She often spoke of her condition of blindness and deafness, and of herself as the "mad Miss M." She evidently could not see or hear, as a general rule; but the sister and nurses believed that she could both see and hear imperfectly at times. It is certain, however, that her sense of touch was very acute; and any slight disturbance of the bedclothes or bed, made her move or stretch her arms in its direction. If she caught hold of any one's hand, she would grasp it and fondle it with both her hands, and press it to her bosom or carry it to her lips, and ask to be kissed. At the same time her face assumed an extremely affectionate, if not erotic, expression. She generally fed herself greedily when food was put into her hands. The tremors and the paralytic condition of the eyes remained unchanged. She never passed her evacuations into the bed. On the 19th a discharge took place for the first time from the right ear.

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[1] This is the effect of the only record as to this matter. I believe, however, the tendon reflexes were examined on subsequent occasions and found present.

During the next month the symptoms, on the whole, remained much the same as they had been. She was still often drowsy; but at times was restless; on the whole, however, much quieter than she had been. She still talked somewhat incoherently at times, and in her slow plaintive manner; and evidently did not always know where she was, or who the persons were about her. She was still blind and deaf, and the movements of the eyes were more or less independent of one another. She remained acutely sensitive, however, and understood many things. Thus, she was still inclined to be affectionate, she still fed herself when food was put into her hands, and if she felt stockings being put on her feet, she knew she was to get up, and she assisted in the process. The tremulous condition of head and neck and arms, and in a less degree of the legs, continued. The discharge from her ear persisted. No further paralysis had been developed; but the weakness of the right seventh, which had been before observed, was still present. Her tongue was clean, her appetite good; she had no sickness; her bowels were regular; she passed her evacuations consciously; her temperature was normal; and she was habitually got out of bed and placed in an armchair some time or other during the day.

On the 21st of May her temperature rose unaccountably to 102·2, and the next day to 103·2. She became, perhaps, somewhat more drowsy than she had been; and the ear that had been discharging presented at the meatus an unhealthy-looking sore. On the 25th it was noticed that she had lost power in her legs; and she moaned a great deal, as if in pain. In the evening she began to tremble violently, in arms, legs, head and neck, and trunk; but on the right side more than on the left. This continued for several hours. The temperature was then between 101 and 102. On the 29th it was observed that the legs remained weak, so that she could not stand; that she choked a good deal when taking food; that the veins of the forehead and inner extremities of the eyelids (especially the latter) were greatly distended; that she seemed in pain, but was very quiet, and that her pulse was 92, very feeble and slightly irregular. She gradually sank from this time, and died at 5.15 on the morning of the 30th. Her temperature, which during the last ten days had varied a good deal, but on the whole had continued high, rose an hour before death to 105·4.

It is noticeable that the patient never complained of giddiness and was never sick; that her evacuations were always passed consciously; that she had no fits, unless the prolonged tremors which occurred a few days before death are to be regarded as a kind of fit; and that she had no pain in her head until shortly before her death.

Post-mortem Examination.—There was slight general congestion of the surface of the brain, with flattening of the convolutions and diminution of the subarachnoid fluid. The lateral ventricles were largely distended with fluid. The parts bounding these cavities were somewhat softened. Originating, apparently, in the valve of Vieussens was a soft, translucent, vascular new-growth, which extended into the neighbouring part of the cerebellum, mainly its middle lobe, and appeared to involve to some extent the corpora quadrigemina, which looked swollen and œdematous. It was adherent to the floor of the fourth ventricle, the ependyma of which was converted into a thick pulpy layer; but it did not appear to have encroached on the subjacent medulla. The growth altogether was perhaps about the size of a bantam-fowl's egg; but the limits between it and the nervous tissue, which it invaded, were very ill-defined. All other parts of the brain were healthy. Cord healthy.

Patches of dura mater, in both anterior fossæ, in both middle fossæ, and in a less degree in both posterior fossæ were the seat of growths identical with that affecting the brain. They formed irregular tracts, were a line or two thick, soft, translucent, slightly adherent to the surface of the brain above, and had caused erosion and honey-combing of the subjacent bone-tissue. Some of the nerves issuing from the skull seemed to have been more or less pressed on by these growths; and it was believed that the affection of the ear observed during life was due to the affection of the dura mater.

The neoplasm appeared to consist of small oval and fusiform cells and blood-vessels.

Thoracic and abdominal viscera all healthy.

CASE III.—*Tubercular mass in third ventricle involving both thalami and followed by tubercular meningitis.—Tremors and rigidity of arms and legs; drowsiness; irritability; optic neuritis; slight facial palsy. Convulsions, coma, and elevation of temperature coming on with the meningitis.—Death.—Autopsy.*

Frederick P., aged 4, was admitted under Mr. Sydney Jones, for scrofulous disease of the left astragalus, which was excised on the 22nd of October, 1878. He went on very well until the 28th of January 1879, when tremulous movements were observed in the left arm and leg. These occurred only during the performance of voluntary movements, and consisted mainly in small to-and-fro movements at the larger joints, resembling those of disseminated sclerosis. There was slight loss of power in both of the affected limbs; but he could grasp with a fair amount of force. No other symptoms whatever, referable to the nervous system, were present. The tremors continued, but it was noticed that they were more severe on some days than on others, and that they ceased during sleep. Early in March tremors first showed themselves in the right hand; and slight spasmodic movements were observed on the left side of the face. All these symptoms continued, with little or no material change, and without the development of any fresh ones, up to the 12th of May, when he left the hospital.

He was re-admitted on the 8th of July. He had rapidly got worse, in respect of his ankle, his nervous symptoms and his general health; and latterly had become drowsy, and passed his evacuations unconsciously.

Face pale; a large distended vein runs transversely across the right upper eyelid. Very sleepy, and indeed seems always asleep, excepting when he is disturbed; and although his appetite is good, and he has no sickness, he never asks for food; but he eats ravenously all that is given him. He constantly falls asleep while taking his meals, and has to be roused for each fresh mouthful. He does not seem to be in pain; but has general hyperæsthesia; is very peevish and irritable; and cries out when contradicted or disturbed. When nurses or others stand at his bedside, he attempts to ward them off with his right hand, and whines "No."

The mouth is natural when at rest, but is much drawn to the right when he laughs or cries, while the right eye becomes partially closed, the left remaining open. No squint; pupils equal, dilated, responding to light. Commencing optic neuritis; veins tortuous, no hæmorrhages. Tongue protruded straight, tremulous. Speech slow, but otherwise natural. No deafness. Head retracted somewhat as he lies. There are rhythmical tremors of the right hand and arm and leg, which are excited only by voluntary movements, and cease during sleep. They are much more pronounced than when he left the hospital. The movements on the left side have ceased; and the limbs have become rigid; the left elbow-, wrist-, and finger-joints are strongly flexed. The finger-nails are digging into the palm; and great pain is caused by the attempt to straighten the fingers. He often rubs or scratches the right side of the face. No dysphagia or vomiting; bowels constipated; motions passed unconsciously. Temperature ranging from 99·4 to 101. Pulse 114. Respirations 24.

During the next three months his condition varied little. He was drowsy; often seemed in pain; and was extremely irritable, crying out when pressed to answer a question. He could still see; but his sight was manifestly impaired, and he would apparently clutch at objects which did not exist. The tremors on the right side continued; and the left arm and leg remained rigid, and for the most part still. Slight tremors, however, were now and then observed in them. There was slight facial paralysis on the left side, but none of the tongue or ocular muscles. He had no vomiting, and no fits; and no evidence of disease in the chest or abdomen was obtained.

On the 8th of October he became worse, and vomited twice; and his temperature next day rose to 103·2.

On the fourteenth the following notes were taken. Has scarcely spoken a word since the 8th; and since the 10th his temperature has varied between 101·8 and 102·8. Yesterday afternoon he seemed fairly well, could see, and recognised his mother. But at 10 P.M. it was observed that his left pupil was much larger than his right, and that he took no notice of anything placed in front of his eyes. At 4 A.M. this morning he got unconscious; the left limbs became extremely rigid and tremulous; he foamed at the mouth; his eyes were directed upwards and to the left, and his eye-lids were blinking. He was still in this condition at 10 A.M. He had not called out, and had taken no food. He remained unconscious during the rest of the day, lying with head strongly retracted, breathing stertorously, and having every few minutes attacks of rigidity, affecting both arms and legs, but limbs of right side mainly. Drops of sweat collected on the face, and the right arm and leg became notably cold; but the temperature of the body rose to 103·2. He was unable to swallow.

Death took place at 4 A.M. on the 15th.

The eyes were examined shortly before death, when the veins at the back of the left were found dilated and tortuous. The right presented recent ulceration of the cornea.

Post-mortem Examination.—Much emaciated; pupils of equal size. Old cicatrix over inner side of left ankle.

Meningeal vessels congested; pia-mater on vertex natural; convolutions flattened. Interpeduncular space, pons, and medulla, opaque and covered with flakes of recent lymph. A few miliary tubercles in membrane at base; and an abundance of them in the right fissure of Sylvius. Much distension of lateral ventricles with clear fluid. The third ventricle was occupied by an irregularly globular tubercular mass, about 1½ inches in diameter, which partly involved both optic thalami. Another small tubercular mass was found on the internal surface of the left occipital tube.

No tubercular or other disease was discovered either in the thoracic or in the abdominal viscera.

Case IV.—*Tubercular mass in left lobe of cerebellum.—Effusion of fluid into ventricles.—Inability to walk; tremors of arms and head; optic neuritis and blindness; frequent epileptiform attacks; headache, &c.—Death.—Autopsy.*

A. G. T., a boy four years of age, came under my care on the 30th of December, 1879. About three months before that time he was suffering from hooping-cough and measles, and then had several fits. About two months before admission the mother noticed that he squinted; the fits also became more frequent, and he began to complain of pain at the back of the head.

He is a plump, healthy-looking child; seems quite sensible; answers questions, but speaks indistinctly, and drawls out his words very slowly. He can move his legs, but is unable to walk, or even to stand. His hands and arms (especially the left) tremble when they are in use, and there is apparently want of co-ordinating power in them. His head and trunk also tremble when he sits up. He has no squint, and the pupils are dilated and equal. He appears to be completely blind. There is no definite paralysis, no impairment of sensation, and all the reflexes are well-marked. The pain in his head comes on in paroxysms, and is referred to the occipital region; and, when he is lifted up, he screams out, and puts his hand to the back of his head. There are no signs of disease in the thoracic or abdominal organs. Tongue clean; appetite good; bowels confined; urine 1020, urates and phosphates, but no albumen.

His symptoms underwent very little change during the next few months. He remained sensible, but spoke slowly and with difficulty; he suffered at times from severe headache, which often made him cry out, but in the intervals he was remarkably good-tempered, and always ready to laugh; the weakness of the legs, and the tremors of his head and neck and arms continued; and he passed his evacuations into the bed; he retained a fair appetite, and had no sickness. He had frequent fits, sometimes several in the day; the period of insensibility varying from two minutes to a quarter of an hour. In some of them he was quite still, save for a little twitching of the eyes and eyelids, and turning in of the thumbs. In the more severe ones, the arms and legs became rigid, the former being raised above the head, the latter being straightened, with toes pointed; but the rigidity was more marked in the right than in the left arm, and the latter generally became limp during the progress of the attack. The fits were occasionally ushered in with a paroxysm of crying. On the 23rd of January his eyes were examined by Mr. Nettleship, who reported that he thought the boy had perception of light with right eye, for he followed the mirror with it, and the pupil acted fairly to light; that the left pupil also acted, but irregularly; and that the last stage of optic neuritis (woolly discs), passing into atrophy, was present in both eyes, without hæmorrhage, or much swelling. His head was large, and it was suspected that it was increasing in size; its measurements were taken therefore early in March and again early in April; but no progressive change was discovered.

At the end of March, or the beginning of April, he began to suffer from drowsiness; and was becoming manifestly more and more feeble-minded; much less inclined to speak, and also much less inclined to laugh. Further, he often had, besides his stronger fits, apparently momentary attacks of loss of consciousness, and the limbs were often rigid. The surface was apt to become flushed. The tremors of the head and neck and arms continued. His appetite became impaired; and he had difficulty in swallowing, more especially solids.

During May he became very quiet, ceased to speak, often refused food, and it was observed that he did not close the left eye so perfectly as the right, and that it presented a slight internal squint. There was no doubt that he was now quite blind; and the pupils, which were equal and dilated, did not respond to light. His face was now without expression. The circulation was very imperfect, and the hands were cold and livid. He had become very thin.

During the next two months he passed mainly a vegetative existence. He lay almost unconscious, occasionally moaning, but never speaking, though even to the last apt to present a flickering smile when tickled. His arms became more or less permanently stiff, and in great measure ceased to tremble.

Late in June it was observed that the left conjunctiva was insensible, and that an ulcer had appeared in the cornea. This proceeded to perforation and prolapse of iris, which took place about the 14th of July; at which time an ulcer had appeared in the right cornea also. It may be added, that early in June some vesicles made their appearance on the cheeks and forehead, which subsequently became pustular, and that one of them reached the size of a shilling. He vomited once or twice during the month of July. His death had been anticipated daily, for weeks before it actually occurred on the 6th of August, and was due apparently to a combination of coma and asthenia.

Post-mortem Examination.—Much emaciated; head very large, anterior fontanelle slightly open.

Surface of brain dry; convolutions flattened. The ventricles were greatly distended, containing about two pints of watery fluid. The brain generally was soft, but otherwise healthy. A small tubercular mass was imbedded in the grey matter of the orbital surface of the right frontal lobe; and another mass of similar nature, and about the size of a Tangerine orange, occupied nearly the whole of the left lobe of the cerebellum. The brain-tissue around was softened, and the part in contact with the tentorium was adherent to it. The part of the left cerebral hemisphere that was in contact with the diseased cerebellum was superficially softened. And the corresponding side of the pons varolii and medulla oblongata were pressed upon and somewhat softened. There was no evidence of implication of the ganglia at the base of the brain. The optic nerves, especially the left, were small. Heart and pericardium healthy. Lungs collapsed below, but otherwise pale and healthy. Glands at root caseous. Chylopoietic viscera healthy. Some pyelitis of both kidneys, with a few small dark-yellow concretions.

CASE V.—*Chronic Hydrocephalus.—Dropsical accumulations in lateral and third ventricles, in fourth ventricle, and in central spinal canal.—Weakness of intellect: stiffness of neck: feeble, tottering gait: epileptiform fits.—Death—Autopsy.*

B. S., a harness-maker, aged 17, was admitted under my care on the 8th of June, 1872.

He was said to have been a healthy lad until about four months ago, when he began to complain of pain in his back when at work, of headache and nausea. Three months ago, while at church, he was seized suddenly with pain in his back and head, and giddiness so severe, that unless he had been supported he would have fallen. From this time the pain in his head was so intense and constant, that he was compelled to give up his employment. He remained in this condition up to three weeks ago, when he had three epileptic fits. On the 3rd of June he had two others, but did not lose consciousness in them; and for a short time subsequently he had no use of his right side. Since his illness he has suffered from constipation; and has occasionally seen double.

He is is a well-grown, but dull, heavy-looking, large-headed youth; apparently slow of intellect and speech; and slow and unsteady in his movements, yet with no definite paralysis. He has no discoverable disease of the thoracic or abdominal organs. Pulse 84. Temperature 98·6. Urine 1008, free from albumen.

While he was under observation in the hospital, the following circumstances were noted. The patient has no paralysis of any of his voluntary muscles, and no affection of his tactile or other senses. And he has complete control over his rectum and bladder. But his neck seems stiff, and he has manifest difficulty in rotating his head from side to side, and upwards and downwards; he walks feebly and unsteadily, and with a shuffling gait. His mental condition is peculiar; and gives the impression of slight congenital imbecility, which, however, is denied by the friends. While in the hospital, he was occasionally noisy at night time.

On the 22nd he had a fit. On the 28th he complained more than he had previously done of headache. During the night of the 29th he was more noisy than he had been; but otherwise seemed much in the same state as usual. Early in the morning of the 30th, he became suddenly livid in the face, and insensible, and died without any convulsions in the course of a few minutes.

Autopsy.—Head 23 inches in circumference externally. Skull very thin, especially near sutures, and distinctly marked by convolutions. Dura mater healthy. Pia mater injected. Surface of brain dry; convolutions flattened, and fissures almost obliterated. The infundibulum was much distended with fluid; and on removing the brain, clear colourless fluid poured away from this part. The basal portion of the brain, from the crura cerebri to (and including) the lamina cinerea, was distended with fluid, and not thicker than paper. The fourth ventricle was also clearly distended with fluid, and its lateral margins formed on either side a saccular projection just behind the crus cerebri.

On laying open the lateral ventricles, they were found to be largely and uniformly dilated. Many ounces of fluid had escaped; but several still remained. The communication between them, and between them and the third ventricle, was very free. No septum-lucidum, properly speaking, could be found; and the fifth ventricle appeared to be represented by a triangular prolongation of the third ventricle forwards. The corpora-striata and optic thalami of the two sides were widely separated by the large and distended third ventricle. The commencement of the iter was quite blocked up by a translucent septum, looking like a portion of the ependyma. The testes and nates and roof of the iter were together much widened, bulged upwards and very thin, evidently from distension of the iter with fluid. The valve of Vieussens, and processus e cerebello ad testes, were also greatly distended and tense, forming a fluctuating tumour which was continuous with the projections observed behind the crura cerebri. The pons itself looked normal. It was clear that the fourth ventricle and iter formed a common cyst, isolated from all other cavities. The substance of the cerebrum, cerebellum, and other parts of the brain, was of normal consistence and structure. The lining-membrane of the ventricles was somewhat thickened.

The medulla oblongata and the first six inches of the spinal canal were much enlarged, leaving scarcely any space for subarachnoid or arachnoid fluid. This was due to dilatation of the central canal, which was full of serous fluid. There was no communication above between this and the fourth ventricle; and below, the central canal became, as in the normal condition, inconspicuous.

There was no inflammation, tubercle, or new growth of any kind. The nerves at the base of the brain appeared all healthy.

The weight of the brain, including the fluid (perhaps an ounce) in the fourth ventricle, was 66½ oz.

All the other viscera were healthy.

A STUDY OF A CASE OF EPILEPSY.

BY CHARLES MERCIER, M.B.

ELIZABETH C., æt. 29. Family history presents no data of importance. Her first fit occurred at the age of nine, and she has had fits at irregular intervals ever since. Her own account is that she knows when the fit is coming on, that is to say, "she can feel it about her," but she cannot tell when it "takes her." She cannot describe the pre-paroxysmal feeling. "It is too horrible." "It is in her chest, and head, and all over her, but in her chest and head most. It lasts an hour, or sometimes two hours." Before witnessing her paroxysms I placed no reliance on this statement, believing that she had much overstated the duration of the aura; but, as will be seen, the actual duration was very much longer than she describes.

During the nine months that she has been under observation, her fits have occurred in batches of from 4 or 5 to 30 or more, the batches being separated by intervals varying from a few hours to as much as three months. The fits and batches of fits spread over this period varied somewhat in minor details, but in their general characters maintained a remarkable uniformity.

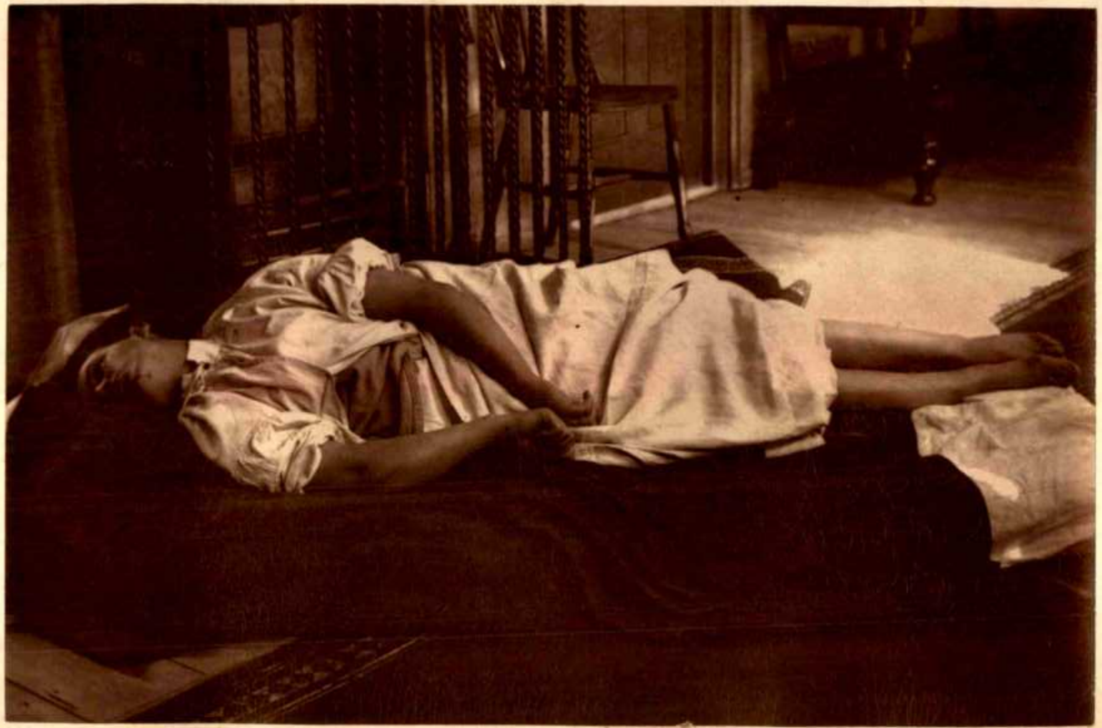
At the outset of every batch of fits the first noticeable occurrence usually is, that the patient becomes captious and irritable, quarrelling with all her neighbours without reasonable provocation. When questioned at this time she answers defiantly and insultingly, in a manner quite alien from her usual demeanour. At the same time, or soon after, she begins to complain of pain, often definitely localised in the side or shoulder-blade, or epigastrium, and later of "horrible feelings" in her head and chest, and all over her. The look of distress that her face has borne hitherto now deepens into an expression of mingled sulky ferocity and anguish; and as time goes on, the latter expression predominates and the former fades out. Next she becomes taciturn, answers impatiently and indistinctly; her articulation is first slovenly, and then decidedly defective. She appears overwhelmed with unutterable languor, her limbs are flaccid and her attitude one of limp helplessness. Her answers come at longer and longer intervals, and at length are elicited only after repeated and urgent questioning; and she no longer looks at you when she speaks. About this time it is noticed that her face has assumed an earthy tint; this is a very marked feature. The complexion is something like that of advanced Bright's disease, but is a deeper and more muddy hue. Her expression becomes more agonised, but has an added and increasing look of vacancy, and at length a definite strabismus appears, marking the period when answers can no longer be obtained, although a trifling movement of the lips or a faint gesture of despair shows that consciousness is not altogether lost. When this stage is reached, the onset of convulsion is not far distant; indeed, it may be said to have already begun, for upon examination at this stage the hands and forearms will be found to be already somewhat rigid, and to have assumed a position approaching that which is characteristic of the paroxysm.

The foregoing series of events is spread over a time that varies on different occasions, but is always considerable. On Oct. 9, 1882, a note was made:—The shadow of the coming fit is apparent for a long time before the fit occurs. Yesterday her eyes had lost their brightness and looked lack-lustre, and a dark areola was noticed round them, five hours before the fit began; and at least three hours before the fit her articulation became worse, she was very languid, and had a feeling which she describes as "horrible." Two hours before the fit, she lay prostrate, and could scarcely be roused to answer, and when she did so, it was found that her articulation, previously fairly distinct, had become almost unintelligible. She could just say that she felt "fitty." On Oct. 29th, at 10.30 A.M., it was noticed that "Her face has an earthy tint, her eyes a sunken appearance, with a dark areola round them, such as is usually noticed before the onset of her fits...she says that she feels 'very indifferent,' and 'as if the fits were coming on,' &c. About 10 P.M. the same night she began to be convulsed. *Dec.* 2.—"After a long intermission of fits, none having occurred since Oct. 29, she began the day before yesterday to complain bitterly of feeling 'fitty,' and of a dreadful feeling in her head. Her articulation at the same time became more imperfect, her eyes sunken, her face earthy, her expression distressed, and after a time a slight squint appeared. She became gradually worse for fully twelve hours, and then began to have fits." The most remarkable instance of prolonged warning is, however, the following. On Feb. 6, 1883, I made a note: "She is less animated than usual this morning, her face has a somewhat dull expression..., &c. It is probable that a fresh batch of fits is coming on." Confirmatory notes were made on the 7th and 8th, and on the afternoon of the 9th a further note: "Her face has a vacant expression, and she squints slightly. When spoken to, she makes an effort to answer, but her words run into one another, and her articulation is so imperfect that it is very difficult to make out what she says," and so forth. It was not until the night following this day that the fits at last occurred; a distinct warning having been observable three clear days and a half before their onset.

At length a definite convulsion occurs, and this event is repeated at intervals of from a few minutes to a few hours, from four or five to twenty or thirty times, when the batch comes to an end.

The several convulsions of a batch are superposed on a condition of rigidity, which begins gradually some time before the first fit, towards the end of the stage of warning; continues without intermission in the intervals between the several fits of a batch; and slowly disappears some time after the last fit is over, leaving a condition in which losses and defects are conspicuous. On one occasion the hands were distinctly rigid at 5 P.M., the last fit having occurred at noon. The position imposed by this rigidity is one of extension and pronation. The head is thrown back, the body is over-extended or opisthotonic, the legs and feet are extended, the arms are by the side of or in front of the trunk, the elbows extended wholly or three quarters, the forearms very strongly pronated, and the hands clenched. The eyelids are moderately open and the eyes to right. The position is well shown in Plates I. and II., save that in the former the extension of the head was artificially diminished, with the intention (unsuccessful) of showing the position of eyes and mouth. The rigidity is greatest in the hands, in which it cannot be overcome by any justifiable amount of force; less in the wrists, still less in the elbows, and not very great in the shoulders. It is less in the legs than in the arms, or neck; the head being retracted with a force approximating to that which immobilises the elbows.

Besides the rigidity, there is another motor phenomenon which is never absent in the inter-paroxysmal stage. At the beginning of every inspiration the left corner of the mouth is suddenly drawn downwards and outwards to an extraordinary extent, the commissure of the lips being at least doubled in length with each such spasm. The rest of the mouth remains unaffected, save in so far as it is disturbed by the dragging of this corner. This movement is mainly due to contraction of the platysma, which is seen to be in violent action, and stands out prominently with each spasm. But the whole jaw moves slightly to the left, and the masseters unquestionably harden at the same time. The spasm of the mouth is invariably synchronous with the respiratory movements. It begins at the same moment as inspiration, continues as long or nearly as long as inspiration lasts, and as the inspiratory movement reaches its height the mouth returns to its symmetrical shape. When, as often happens, inspiration is uniform—when its evenness is interrupted by slight checks, such as may be noticed in the long-drawn sighs of a sobbing child, a corresponding wavering occurs in the spasm of the mouth. With expiration, the lips are puffed outward and vibrate with a purring noise. At the end of expiration there is an interval of quiescence, which is often considerably prolonged, before the inspiratory movement begins; and during this interval the mouth is quiescent. Then the cycle begins again. These movements I have witnessed several scores of times, and have



Woodburytype.

PLATE I.

never seen any variation from the order and combination described. I have notes of another case of convulsion in which an analogous association of spasm with the respiratory movements occurred. In that case also the spasm so associated occurred in the ebb intervening between two tides of convulsion. The spasm took the form of a couple of twitches, —two shocks of "simple contraction"—separated by a brief interval and affecting simultaneously the face, head, arms, and legs. This pair of contractions occurred invariably at the end of every expiratory act,—at the beginning of the pause which precedes inspiration in the slow breathing of coma, and was observed more than one hundred times.

Returning to the case of Elizabeth C.; on one occasion a third motor abnormity was noticed in the inter-paroxysmal period of the convulsions. After a free interval of two months she had had a batch of fits extending over twenty-four hours; then an interval of twelve hours, and then a single fit. After this fit, she was lying on her back, the arms, legs, and neck being rigid as usual. Breathing was slow, deep and laboured, the mouth drawn in inspiration, as above described, and the lips puffing in a flaccid manner that showed deep coma. A rather unusual amount of movement of the chest in respiration led to further examination, and it was then found that the diaphragm was not acting. There was paralysis, total or almost total, of the diaphragm. While my hands were placed on abdomen and chest, for the purpose of examining this matter, I felt a subdued quivering beneath them, and this gradually increased, until at length she was shaking all over with a fine tremor of about 240 to the minute, like a person in a rigor, or shuddering with cold. There was no marked cutis anserina, and the condition of the pupils was not observed. The movement appeared to begin and be most pronounced in the thighs and about the pelvis; but it affected the whole body, as far as could be observed; and when the left forearm, which was not visibly tremulous, was grasped, the muscles therein were found to be quivering in unison with those of the legs. Passive movement increased the tremor, which continued for fully ten minutes, until a fit came on, but did not increase and merge into the fit. In connection with this occurrence it should be mentioned that on one occasion, after a paroxysm, there was tremor of the right arm "as if from fatigue."

The paroxysms of convulsion that are superposed on, or grow out of, this rigidity, are much alike in general character, although they vary a good deal in minor details. In all the spasm is of tonic quality. The movements are, as a rule, slow, flowing and continuous; or if there is a twitching, it has never the electric suddenness of clonic spasm, and the separate simple contractions are never perceptible. The onset is always deliberate. The spasm always begins in the periphery and spreads towards the trunk. It always begins bilaterally, and remains so; but its invasion is rarely quite simultaneous on the two sides, and there is always a preponderance of the spasm on one side,—the right being the first and most effected. Shortly after the onset of the fit, she rolls over to the right and lies on her face, in the attitude given in Plate III. At this time the thorax is locked in rigid spasm, the face is extremely livid, but is not much distorted, and in the otherwise rigid body various large and general movements occur. Subsidence is never by clonus; but the cessation of the spasm of the glottis is announced by a groaning cry, and as the pent-up air is at last expired, a general relaxation occurs, and the body subsides into an attitude of comparative repose; but, on examination, this relaxation is found to have been only partial, the rigidity already described remaining.

The origin of the convulsion, though always peripheral, never, that is, beginning in the bilaterally acting muscles, is yet variable; or rather it would be more correct to say that it begins almost simultaneously in eyes, mouth, and hands, but that the simultaneity is not exact, and that which of these parts shall have a momentary precedence is a quasi-accidental matter. In one paroxysm the first thing noticed is an increasing rapidity of those twitches of the left corner of the mouth that have been described, respiration at the same time becoming imperceptible. Other fits begin by turning of the eyes; but in this case again the ill-defined localisation of the onset is seen in the fact that the first movement of the eyes is often to the left, and not until after a few moments of wandering do they take up their position to the right. The movement of

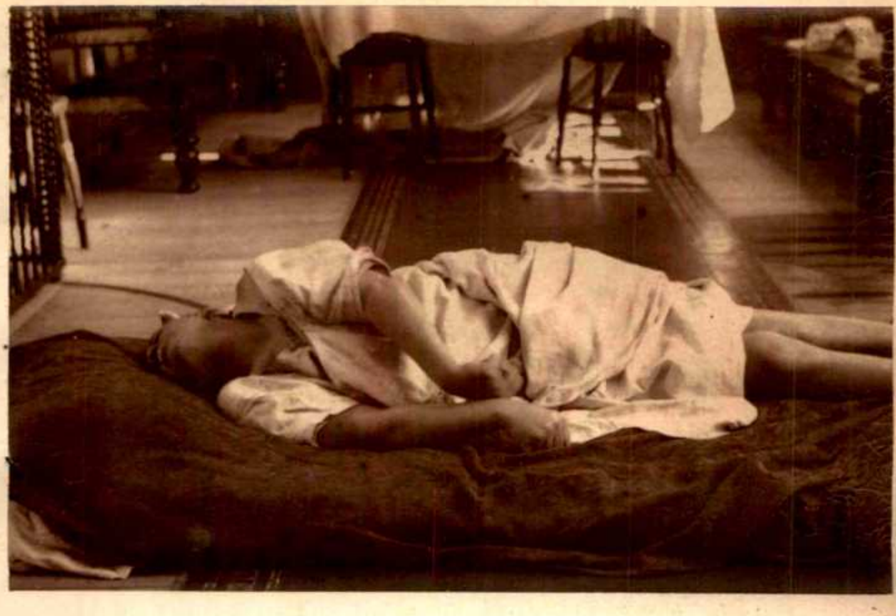


PLATE II.

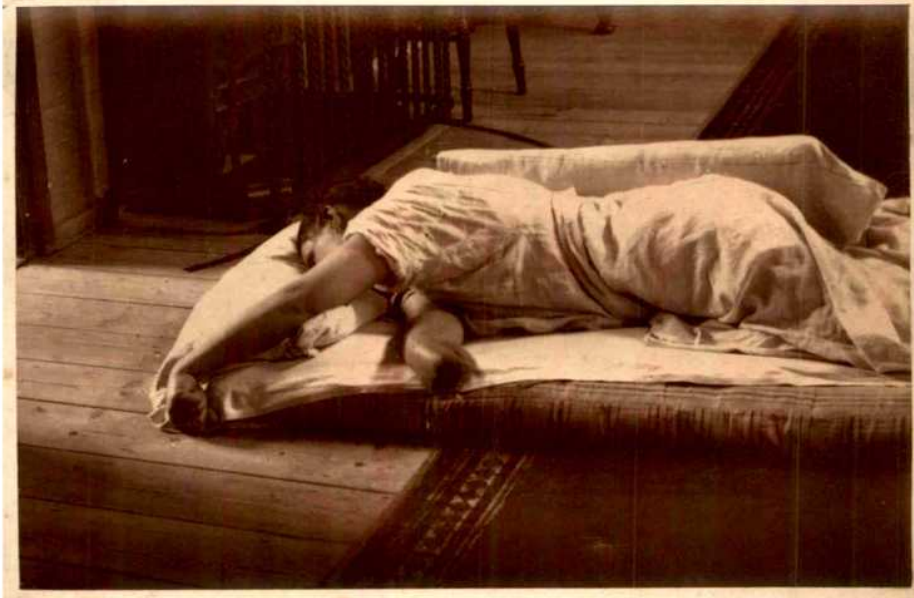


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PLATE III.

the hands at the outset of the fit is not, as is usual, a gradual closing up into a knot of spasm, for the hand is already rigid, but a to-and-fro movement of pro- and supination (pronation being the active element), which is at first about five or six to the minute, and not very sudden, and becomes more and more rapid, until it ends as continuous and extreme pronation.

While there is a general bilateralness of the convulsion, yet the preponderance of the spasm is always on the right. However the eyes may be at first directed, they soon turn to the right, and remain so. The right hand is often affected before, always more than, the left. In every fit she invariably, however she may be lying, turns over and lies on her face, the rotation being always from left to right. Once this attitude is attained, no further one-sidedness of the movements is apparent; but that the right suffers most is shown by the enduring paralysis of this arm and leg, the left recovering rapidly.[1]

At the height of the fit, when the prone position is complete, large general movements of the whole body occur. These often take the form of violent and extensive to-and-fro movements of the pelvis. On several occasions there were very definite alternate movements of the arms and legs,—movements as of quadrupedal locomotion, which, taken in association with the prone position, were very remarkable. They vividly reminded me of the movements of a choreic dog.

Plate III. is an instantaneous photograph taken on one of these occasions. On other occasions the movements were tumultuous tossings about, of indefinite and indescribable character. In every case, however, the electric suddenness of clonic spasm is conspicuously absent. The force of the movements is amazing.

The recovery from the state of exhaustion and coma in which she is left by the last fit of a batch is at least as gradual and prolonged as the onset of the attack; two, three, or more days often elapsing before consciousness can be said to be fully regained; and even when she has recovered in this respect, various sequelæ are found to remain behind. When she first came under notice, in June 1882, the right leg was weaker than the left, and this weakness increased after each batch of fits. After a prolonged attack on Oct. 1 and 5, the right leg remained completely paralysed and anæsthetic. On Oct. 11, after another attack, the right arm remained paralysed, anæsthetic, and rigid in the position of the late rigidity of hemiplegia. This condition remained nearly stationary for six months, and has only lately improved. The most remarkable of the post-epileptic losses, was, however, the following.

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[1] While this is in the press, she has had for the first time a batch of fits in which she turned to the left, and the left arm remained paralysed and rigid after recovery.

After a succession of batches of unusually numerous and severe fits at the end of August 1882, her recovery was very slow. Even on the third and fourth days she appeared dull, heavy, and stupid, and responded to questions only by faint and impatient gestures. As she gradually regained her intelligent expression and her consciousness, she still continued to express herself by signs alone, and it became manifest that she had lost verbal utterance. Her inability to utter a single word, coupled with her evident distress, and the manifest efforts that she made to speak, was at first supposed to be aphasia; but it very soon appeared that she was quite able to write, and to express herself fluently and correctly in writing. The loss was of articulation merely. In the intervals of her convulsions she improved; but each fresh attack threw her back, and reduced her to a more or less totally inarticulate condition, the defect extending altogether over about three months, at the end of which time a long freedom from convulsions allowed of her complete recovery. After fits subsequent to this recovery, the return of articulation has been delayed beyond the return of consciousness, but no prolonged articulatory defect has existed since.

The following note describes her condition at the lowest ebb of this special loss; at that period after the paroxysm when consciousness appeared to have completely returned, but the defect of articulation was still extreme. *Sept.* 15.—"She sits up in bed, looking about her intelligently, and showing much interest in what is going on. She smiles, showing no paralysis of face; she cannot whistle, never having acquired this accomplishment, but she readily pouts her lips into the appropriate position. She kisses (her own hand) quite perfectly. She puts out her tongue when told, and performs adroitly every movement that is indicated to her. She makes, without the least perceptible defect, the clicking noise familiar to coachmen, and utters rapidly the 'tut tut' of impatience. She can also clear her throat with a sonorous hawk. But although she speaks fluently, she cannot articulate a single word. Her utterance consists of only one vowel, without any consonants at all. Imagine every vowel sound in every word to be replaced by the single sound *ah*, or *ăh*. Imagine every consonant to be discarded, and replaced by a mere momentary break in the utterance—a break formed, not by any movement of lips, tongue, or fauces, but apparently by a closure, or partial closure, of the glottis, and a respiratory check. Imagine the words thus degraded to be uttered rapidly, vivaciously, with a very varied cadence and abundance of vocal expression; and you have the utterance presented. While the lips, tongue, jaw, palate, and fauces, appear to be absolutely passive, and to take no part in articulation, her words, if words they can be called, can often be recognised, and her meaning apprehended, from the rhythm of the utterance, taken in association with the cadence, and regard being had to the probable nature of the propositions that she attempts to express." The various consonants and vowel sounds were carefully gone over, the general result being that labials were sounded worst, dentals more successfully, and gutturals, after several efforts, fairly well. All the vowel-sounds were first uttered as *ah*, but after a good deal of practice, she could, after beginning the required sound as *ah*, subsequently modify it into the sound required. Thus *ee* was uttered as *ah-ee*, *o* as *ah-o*, &c. At this time she could write quite fluently, but with curious omissions of words, a defect that was not aphasic, but peculiar. For instance, I had been speaking of sending her back, and she wrote: "I don't want you—me back," the words "to send" being omitted. Subsequently she wrote, "You don't feel—but I do. I never be like this. Any one would have—told me this six months ago." Her meaning doubtless was, "You don't [know what I] feel, but I do. I never [would have believed that I should] be like this, [if] any one would have told me this six months ago." After some days, however, this defect disappeared, and she wrote her history at length, quite intelligibly, while she was totally inarticulate. While writing, she made grimaces (she is unused to writing), but did not form the words with her lips.

It is unnecessary to record the fluctuations that from time to time occurred in her power of articulating, but it may be said generally that the vowels always returned before the consonants, that the sound *ah*, was always the most persistent form of utterance, and that the consonants showed no regularity in the order in which they were regained. Labials returned sometimes after the other consonants, sometimes first of all. Occasionally she could utter a sound, especially a dental sound, at the beginning of a word, when she could not utter it as a terminal. On one occasion she totally lost even the rudimentary sound that usually represented her articulation, and while she indicated her appreciation of what was said to her by doing as she was told, she failed to produce even the remnant of a reply.

On the morning of the 9th of October a note was made that she had almost entirely regained her articulation, and details in proof of this statement were taken. In the afternoon, however, she displayed a defect of more complex character which was not present in the morning. She was somewhat aphasic. "The words that she does utter are better said than usual, but before they issue there is a long pause, and an appearance of great effort; and there is a violent expiration in getting them out. Some words fail to be uttered even in caricature, and efforts to utter them end with the shake of the head and the despairing gesture common in aphasia. She has always been very voluble in her own jargon, but now is silent. On being asked a question, she says 'I—I—' and then a despairing gesture. Some words are uttered after many repetitions of the initial letter and with exaggerated emphasis, as in stammering. She appears to think for the right word, and to be unable to find it, like an aphasic. She understands clearly, however, a rather intricate pun." On the evening of this day she had several convulsions.

Shortly after this the right hand remained, after some severe convulsions, rigid, and therefore useless, and on being asked to write with her left hand, she wrote fluently in mirror-writing. It appeared, however, that she had learnt this before. Although not herself left-handed, her maternal grandmother was so, and her own mother was ambidextrous.

It was noticeable that at a time when her articulation was quite normal at the beginning of a conversation, it would fail towards the end, when she got fatigued.

Comments.

In this remarkable and instructive case, of which the main features only are enumerated in the foregoing account, the points that most call for consideration are three; the prolonged warning, the character of the convulsions, and the articulatory defect.

Taking first the articulatory defect, and first of all the intrinsic relations of this defect, stress must be laid upon the fact that, speaking generally, and allowing for aberrations in detail, the occurrences followed the law laid down by Dr. Hughlings Jackson—the special arrangements suffered most and returned latest, and the general arrangements suffered least and recovered earliest. That the vowel-sounds have a wider generality than the consonants is sufficiently indicated by the fact that the former are capable of standing alone as integral articulatory wholes, while the latter are merely accessory and have scarcely an existence, save as limiting a vowel-sound. The first articulatory element to appear in the infant is simple phonation—an undifferentiated vowel-sound. The first element to become firmly established in civilised races is the broad *a*, whose fundamental quality is shown by its forming the staple of the first articulate utterances, "da-da" "pa-pa," &c. Not until the arrangements for this sound have become to some extent organised, do the other vowel-sounds appear.[1] That consonants appear later still is seen not only in the developing child, in whom some of them often remain undeveloped for several years, but in the fact that the lower animals can scarcely be said to possess any consonants. Now in the case here recorded, the vowels had completely returned at a time when the consonants were still defective; the former were partly retained at a time when the latter was wholly lost; and of all the elements of articulation the broad *â* was the first to be recovered; the other vowel sounds being subsequently acquired by gradual modifications of it. As to the consonants, there was no constant order of recovery, but neither is there any constant order of acquirement, nor could any consonant be said to be more highly special than the rest. Occasionally she was able to articulate a word perfectly in its appropriate place in a sentence, although she failed to utter it when told to do so. The corresponding defect, which is common in aphasia, has been pointed out by Dr. Jackson to be the loss of the most voluntary and special mode of using the word.

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[1] That the early appearance of this sound is not peculiar to our race, and not, therefore, a quality that has been lately acquired, is shown by the facts of comparative philology. For I find that out of 417 languages, the predominating vowel-sound in the word for "father" is *â* or *a* in no fewer than 315, or more than three-fourths; while out of 316 languages the predominating vowel-sound in the

As to the extrinsic relations of the loss of articulations—its relations with the condition of the neighbouring faculties—the most notable circumstance was the isolation of the defect. Not merely was there no paralysis of the muscles involved, not merely were all the other buccal, lingual, &c., movements normal, but at a time when speech, as indicated by writing, was perfect, and cadence was not in the least impaired, articulation was altogether lost. Are we then to conclude that the faculty of articulation is localised in a region of the grey matter distinct from those appropriated to speech and cadence? The dictum against multiplying causes should render us very re-

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word for "mother" is the same in 190, or nearly two-thirds. As it is often difficult to infer the correct sound from written words, I left out of this computation all cases in which the sound appeared doubtful. Taking the facts in another way, the fundamental character of the sound becomes still more apparent, for out of 417 languages there are but 42, or one-tenth, in which the vowel *a* is not used in the formation in English writing of the word for "father;" and out of 395 languages there are only 72, or between one-fifth and one-sixth, in which the same vowel is not used in the formation of the word for "mother." Had languages of Aryan stock alone been considered, the proportion would no doubt have been greater. The facts were extracted from Professor Buschmann's tables.

luctant to think so. Yet many facts indicate a certain degree of solution of the common matrix from which they have unquestionably been differentiated, and that they cannot be looked on severally as mere grades in the larger faculty of language appears certain. Were they so, not only should all be disordered concomitantly, but, according to the law above quoted, speech should always be affected first and most, articulation less, and cadence least. But in paralysis agitans the very reverse obtains. Cadence is lost, articulation is slightly affected and speech is normal. Moreover, the facts that a person can read fluently the words of a language that he does not understand, and that, as in the case related by Coleridge, a person in delirium can repeat pages of a foreign tongue of which she does not understand one word, appear to demonstrate beyond question that articulation is largely separable from speech, and that the two may therefore be disordered independently. In the present case, although articulation suffered by far the more severely, yet there is evidence that in the onset speech was affected first. Aphasia is the loss of the physiological activity of a certain tract of the highest nervous arrangements, and when this tract alone is thrown out of action, the defect shows out conspicuously by contrast with the unimpaired activity of the surrounding nervous arrangements. If we look on the physiological basis of consciousness as a continuum of activity throughout all the highest nervous arrangements, then if this activity be progressively diminished in all these centres simultaneously, there will be a progressive degradation of consciousness in all its forms; and the defect of any individual faculty will be merged in the general loss, and may be overlooked. In this way the aphasia that was unquestionably present in the prolonged stage of invasion was rendered inconspicuous, the loss of a part being drowned in the loss of the whole. On one occasion, however, in the gradual degradation of processes that occurred before the fits, the defect in the language series was sufficiently in advance of that in the others to be separately recognisable, and accordingly a condition of unmistakable aphasia resulted, as detailed in the note of the 19th of October. Granting, however, that speech was first affected, it must nevertheless be admitted that it was not affected most severely, and it certainly recovered long before articulation; so that while there is evidence that the discharge began according to rule in the highest centres, yet it must during the convulsive stage have assumed an overwhelming preponderance in centres comparatively low in grade, and that this was so is indicated by all the localising characters of the convulsions themselves.

To recapitulate these characters:—The spasm was of tonic quality. By tonic spasm I mean spasm in which the constituent "simple contractions" never become discrete and separately perceptible, but are fused together, as in this case, into waves of gradual rise, progress, and decline. Next, the spasm was bilateral and approximately symmetrical. Beginning deliberately, it was seen to invade both arms independently and almost simultaneously, and to travel centripetally from the hands up to the trunk; the attitude produced was one of universal extension, and in the forearms of pronation also, and this attitude was the same in both sides. If we remember the characters of Jacksonian epilepsy—convulsions which are clonic from the outset, which always begin unilaterally, and commonly in one thumb and finger, which often remain throughout conspicuously unilateral, which never produce a symmetrical attitude on the two sides, in which flexion invariably and everywhere preponderates, and the forearms are commonly supinated, we shall see that they are in almost every respect the reverse of the convulsions in this case, the only common feature being the centripetal march. And the convulsions of Jacksonian epilepsy are, we know, due to discharging lesions of the cortex of the cerebrum. Hence the inference is valid that wherever the discharging lesion is in this case, it is not in the cerebral cortex. Again: the excessive discharge of a region of highly elaborated centres is necessarily attended by the abolition of the function of the parts represented. When a cerebral region is so discharging, the several movements that should normally occur separately in orderly succession are all jammed into the same moment—all strive to occur at once—and the result is a chaos of spasm. But when a region represents but a single undifferentiated movement, the discharge of that region, however excessive, however rapid, can cause only that one movement, which is still therefore a co-ordinated movement, though it is excessive in degree. For instance, when the vaso-motor region so discharges, the normal movement—contraction of the arteries—still takes place, as we see in a rigor, although of course it is excessive in amount. Applying this consideration to the case of centres intermediate in grade between the most elaborate and the simplest, it appears that we can judge approximately of the grade of a centre that is discharging excessively, by the amount of co-ordination that exists in the resulting movement. Now in the present case the outcome of the discharge was not a mere chaos of rigid spasm. There was a distinct co-ordination of a simple kind remaining, as was evidenced in the movements described as resembling those of quadrupedal locomotion, in which all four limbs were concerned, and moved in times definitely relating to each other.

(I have seen two other cases in which definitely co-ordinated movements occurred during the actual progress of a convulsion. In both cases the convulsions affected first and most the bilaterally-acting muscles, and the attitude produced was one of general extension. In one case violent and repeated sneezing (note the connection of the spasm with the respiratory function) occurred both at the outset and at the close of the fit. In the other the patient, while at the height of the fit, used to bite the back of her hand. The first of these movements is so deeply organised as to have become reflex; and the second was no doubt a caricature of the action of carrying food to the mouth—also a movement, which, though much more elaborate than that of sneezing, is yet very deeply organised in the race.)

The tonic quality of the spasm, the continuous duration over many hours, the symmetrical distribution, the attitude of extension, and the rotation which was so prominent a feature in every fit, and which never failed to occur, all powerfully suggest that the spasm was of cerebellar origin; but, on the other hand, the centripetal march of the spasm seems to me to outweigh all this evidence, and other circumstances reinforce this objection and appear definitely to negative such a supposition. The occurrence of anæsthesia is not perhaps of much weight, since it may have been due to exhaustion of some lower tract to which the spasm had extended. More consideration is due to the fact that the position of the arms in post-hemiplegic contracture and in tetanus, both of which are believed by Dr. Hughlings Jackson to be due to cerebellar influx, is one of flexion and supination, while here the arms were extended and pronated. But the most fatal objection is the existence at the height of the convulsion of those definitely co-ordinated movements already referred to as having a distinct, if a caricatured, resemblance to those of quadrupedal progression. The tendency of recent thought as to the functions of cerebrum and cerebellum is more and more towards the view that neither of these organs alone can affect a co-ordinated movement. The cerebrum is powerless to bring about such a movement in the absence of the underlying tone pre-existing in the muscles by virtue of their cerebellar supply. The cerebellar discharge alone can produce merely a static and enduring contraction which must be broken up by the interrupted discharge from the cerebrum before a co-ordinated movement can occur.

On the whole, then, it appears very unlikely that the paroxysms of convulsion in this case can have been directly due to discharge either of cerebral or of cerebellar regions.

If then the discharge was neither cerebral nor cerebellar, what was its origin? The simultaneous invasion of both hands implies that the discharging region was below the great bifurcation of the pyramidal tract, and the balance of the evidence favours the view that the main focus was at the upper part of the pons varolii. The very low grade of this region, compared with that of the cerebral and cerebellar cortex, and the fact that fibres from both these latter regions are here in close proximity, will account for the slight co-ordination that existed in the excessive discharge. The nearness of the middle peduncle of the cerebellum and its extension in the transverse fibres of the pons will account for the rotation that occurred. The proximity of the respiratory tract will account for the definite connection with respiration, and the existence of anæsthesia and of the articulatory defect are easily intelligible if we admit the main focus of the discharge to have been in this region.

Seeing that the hypothesis of a pontic origin will account for the main features of the attack, let us ask if there are any features with which it is inconsistent. There is one which appears to offer a fatal objection. I have endeavoured to show in previous numbers of "BRAIN" that a discharge never does and never can begin, save in the highest centre of a series; and the centres here discharging were admittedly of very low grade. Discharge of such a region from general causes is only possible on the assumption that all the superposed centres were non-existent, as far as function is concerned—that they had all been previously discharged. Yet there were no convulsions antecedent to those of the lower region, to indicate previous exhaustive discharge of the higher. Nevertheless I think there is very cogent evidence of a different kind to show that such a discharge had taken place, and that all the higher centres were for the time being *hors de combat.* It is quite true that there had been no convulsion representing discharge of the cerebral centres; but for hours and days before the convulsion, which I suppose to have been due to discharge in the pons, there was a gradual and progressive loss of all the functions of the higher centres. There was hebetude gradually deepening into unconsciousness; there was languor gradually increasing into helplessness; there was weakness gradually increasing into paralysis; and, perhaps most significant of all, there was that indescribable feeling of horrible depression which, as we see in melancholia, accompanies extreme feebleness of the cerebral energising. All these occurrences point with converging indication to a gradual obliteration of the cerebral functions, progressing from the highest downwards; and it is difficult to see what possible origin this obliteration could have, other than a gradual discharge. Yet there was no convulsion to indicate such discharge. Guarding myself against too dogmatic an interpretation, I would ask if this absence of convulsion may not fairly be ascribed to the extremely deliberate and gradual escape of the liberated energy? That it was very deliberate is shown by the fact that it took many hours and even days to reach the same degree that is reached in an ordinary epileptic fit in a few seconds. That it was very gradual every observation showed, for no sign of definite stages was ever observed. No fact in nerve-muscle physiology is better established than that the contraction of a muscle depends primarily on the suddenness with which the stimulus is applied to the nerve. A tap, a cut, a shock of electricity—any stimulus whatever that is suddenly or rapidly applied to the nerve, brings about a contraction of the muscle. But a pressure very deliberately applied and very gradually increasing may be added, until the nerve is crushed to pulp, without inducing the smallest movement of the muscle. A continuous galvanic current may pass through the nerve for hours and days without inducing a contraction, save when it is applied and removed; and further, although an increase in the strength of the current is at once followed by a contraction, yet if the increase be very cautiously and gradually made, the contraction may be avoided. It does not, therefore, appear to be claiming too much to suppose that the whole available surplus of energy in the cerebral centres may ooze gradually away without giving rise to any convulsive phenomena whatever; and may thus leave the lower centres uncontrolled and free to indulge in the most rampant excesses of action.

There is a little circumstance which appears to me to corroborate this view. In ordinary cerebral convulsions of deliberate onset we can often cut short the fit by administering chloroform at an early stage. Chloroform, as we know, ablates the function—renders impossible the discharge—of the cerebral centres in their order from above downwards; and if we can thus arrest a discharge, we can of course arrest its extension. But if, as supposed, the convulsion in this case occurred from the excessive discharge of low grade centres, due to removal of the control of the centres above them, then anything that accelerated the removal of this control should accelerate the onset of the paroxysm; and this unquestionably was so. On one occasion, while she was in the stage of "warning," the patient's complaints of her horrible feelings were so bitter, and her sufferings were evidently so severe, that I attempted to give her relief by the administration of chloroform, hoping thus not only to relieve her suffering but perhaps to ward off the fit. To my surprise, she had no sooner taken a few whiffs than the convulsion began. I say to my surprise, for the very fact that she was able to complain showed that she had not yet nearly reached the stage at which convulsion usually occurred.

The importance of this view here advocated is this, that if it be adopted, it may lead to the explanation of those numerous cases in which maniacal attacks precede the fits in cases of epilepsy,—cases which have always been obstacles in the way of adopting the hypothesis of Loss of Control. In this very case the hebetude and languor that unquestionably indicate a decline of cerebral energising, be the cause of that decline what it may, are not the first occurrences in the attack. They are commonly preceded, as was stated in the notes, I may say, long before this explanation occurred to me, by a period in which her character and disposition are altered. She becomes irritable, quarrelsome, insulting and foul-mouthed; qualities so alien from her usual conduct, that a lady who has frequently visited her, but not at these times, positively refused to believe that she behaved so. She is not actually violent; but this I believe to be simply because she is bedridden, on account of her paralysis, and therefore physically incapacitated from violent conduct; but she uses horrible threats, and this is incipient violence. On one occasion she was acutely maniacal, but this was in the interval between two batches of fits, and may be looked on as post-epileptic as truly as pre-epileptic. Leaving this attack out of consideration, there remains the fact that the very first indication of an approaching fit is a degradation of the disposition, a defect of self-control, and a heightened action of the lower and more brutal nature, similar to that which is common in drunkenness, and which is generally understood to indicate the loss of the highest of all the nervous arrangements.

Not only will the proposed explanation account for pre-epileptic mania, but it may also indicate a relationship between the insanity that occurs in epileptics and insanity that is not manifestly so associated. For if we imagine the progressive degradation that occurred during the "warning" of the paroxysms to be arrested at a very early stage,—if we imagine it to go no further than that period when her disposition was so changed by what, following Dr. Hughlings Jackson, I suppose to be the emerging into undue prominence of her lower nature, consequent on the loss by epileptic discharge of the highest of all nervous arrangements,—and if we suppose the periodicity of the attack to be unchanged, the case will be one of recurrent mania, and no one would dream of referring it to an epileptic origin. Imagine the process to have gone a little further, and to be arrested at the stage of languor, listlessness, hebetude and intense mental depression,—depression so great that on more than one occasion she has attempted suicide,—imagine this condition to endure instead of sinking to a lower degree, and the case is one of acute melancholia. Let me not be understood to mean that every case of melancholia, or of recurrent mania, is of epileptic origin; all that is contended for is, that when stages in a malady are found to correspond with permanent states existing elsewhere, we are justified in supposing that the conditions underlying these stages are similar to the conditions underlying the permanent states; and that when we know something of the first set of conditions and nothing of the second, it is a tangible advantage to be able to assimilate the two.

There are abundance of other points of interest in this case that might be treated of, did space permit, such as the interparoxysmal spasm of the mouth, its rhythmical character and association with respiration, and the universal tremor that occurred on one occasion, occurrences that might all be shown to harmonise with the explanation offered; but I trust enough has been said, not indeed to secure acceptance of the views proposed, but, what is far more important, to show the great value and importance of systematically recording the phenomena of convulsion. To this endeavour we have in photography an aid whose importance can scarcely be exaggerated. To write down with any approach to completeness the successive phases of a convulsion as they pass before the eye is impossible, and even dictation to a shorthand writer is a very imperfect way of getting a record, so great are the difficulties both of observation and description. But when twenty-four successive attitudes of a galloping horse can be photographed within the limits of a single stride, there ought to be no difficulty in obtaining a satisfactory record of the successive phases of a convulsion; and now that dry-plate photography is so easy and so cheap, an apparatus ought surely to form part of the armamentarium of every hospital.

ON A METHOD OF DEMONSTRATING THE CONNECTIONS OF THE BRAIN IN HEALTH AND DISEASE.

BY D. J. HAMILTON, M.B., F.R.C.S.E., F.R.S.E.

Professor of Pathological Anatomy (SIR ERASMUS WILSON *Chair*) *Aberdeen University.*

ONE of the great wants that must have been felt by those who have worked at the anatomy, normal and pathological, of the brain is that we do not possess any ready means of preparing the organ so as to show the course of the fibres, and their relationship to the different grey centres. We have had plenty of mere microscopic histology, but as to the more remote connections of one part of the organ with another, we know as yet comparatively little with accuracy. In order to see these, it is necessary to examine the brain in thin *entire* sections cut in different planes; for when one part is separated from the other, the various tracts of fibres are interrupted in their continuity, and the very information which we most desire to have fails us. I have now for many years been working on this subject, and have at last arrived at a process by which not only the brain may be cut in its entirety in any direction, but by which the fibres are rendered almost diagrammatically evident. The results which I have obtained by the process I hope to publish shortly, but, in the meantime, as it may be acceptable to those engaged in what is one of the most interesting branches of pathology, I beg to describe the process which I employ.

Hardening of the Brain.—It is of no use trying to investigate the course of the brain fibres unless the organ has been hardened completely into the centre. Any softening which may take place in the process of hardening, from the hardening fluid not finding free entrance to the central parts, entirely unfits it for any further manipulation. The employment of spirit for the purpose of hardening cannot be recommended, as it causes shrinking and produces consequent deformity. The hardening solution which I employ is a mixture of

Müller's Fluid, 3 parts.
Spirit. Vini Meth., 1 part.

These are mixed, filtered through linen, and allowed to cool before being used. The mixture should never be made until it is going to be employed, and should be kept in the dark.

The brain must be injected with this on three successive days from one vertebral and the two carotids. Unless it is injected, and thoroughly injected, it will never harden. Between fifty and sixty ounces should be injected on the first day, and from thirty to forty on the second and third days. The injection should be driven in with a large syringe, and considerable pressure is required to enable it to penetrate to deep parts. The organ swells to quite double its original bulk when the proper quantity has been forced in. The membranes are sometimes so stretched that they rupture, and the brain may become torn. This must be avoided by not driving in too much liquid at a time. The great danger, however, in injecting is that the points of the canulæ are accidentally driven through the coats of the vessel. This can be avoided by attaching a pliable india-rubber tube about a foot long to each canula, so that the motion of the hand or syringe does not act so directly on its point. When one tube is injected it can be tied up to some support, and thus any tearing of the brain substance by the cross-bar or other part of the apparatus is avoided. The utmost care must be taken in this matter, as a rupture of the internal carotid renders the difficulty of thoroughly injecting the organ much greater.

It is not necessary to inject it while in the body; it injects much more fully when removed. It must, however, be removed with caution, as any tearing, of course, interferes with its perfect distension. The internal carotids should be cut off as long as possible. In removing the calvaria, the outer table should alone be sawn through, the inner plate being broken with a chisel and mallet. It should be kept in a large round earthenware jar, filled with the same mixture as that which was injected. No padding is necessary—it tends to cause deformity. The best result is obtained by simply having an abundant excess of hardening fluid, and allowing the brain to lie freely in it. The natural shape of the organ is thus preserved.

In a month from the last injection—that is, from the third day after removal—it can be taken out of the fluid and cut into entire slices one inch thick with a long thin knife, in the particular direction in which it is intended to make the sections. These should lie in the fluid for another fortnight. If the injection has run properly, the parts internally should be tough and thoroughly impregnated with the Müller's fluid mixture. The great test of the injection having been successful is that the septum lucidum and surrounding parts are entire and perfectly hardened. The hardening has now to be completed by placing the segments in pure methylated spirit. This can be done without shrinking occurring when the brain tissue is fixed by means of the former fluid. I generally allow it to lie for two weeks in the spirit, and care should be taken that the segments are separated from each other by pieces of linen or cotton-cloth, or some other absorbent substance.

Colouring of the Convolutions.—When thin sections are made of the brain, it is almost impossible to tell what convolutions are immediately under observation. In order to mark these out, it is necessary to colour them differently with some material which will adhere to the pia mater or arachnoid membrane. The mixture which I have found most serviceable is gold-size and different ordinary pigments in powder. They are to be ground up in a mortar together, and applied by means of a camel's-hair brush, the colouring matter being well rubbed into the membranes, so as to take a firm hold. The best stage for putting the colour on is when the segments are removed from the methylated spirit. They should be all re-adjusted, so as to see exactly where the convolutions lie, and should be partially dried outside by exposure for two or three hours before being painted. A good thick layer of the paint is necessary in order to be seen in a thin section. The colours used should be such as are not decomposed by potash. Potash has to be employed later on, and certain colours fade by its action. The best that I have found are vermilion, lamp-black, and oxide of zinc. They should be applied individually to convolutions as far apart as possible; and by leaving some uncoloured and by colouring others, there can be no uncertainty as to the part of the cortex concerned. A key to the order in which they are coloured should be kept for future reference, and it is well to colour all brains alike to prevent confusion. One side only should be coloured, and always the same side. I generally employ the right. The particular side of the brain is thus indicated, whatever the surface may be that is uppermost. It is a very good method to follow in the investigation of the spinal cord as well.

Preparation of the Brain for Cutting.—The only method by which the sections I am about to describe can be cut is by freezing. Any one who has seen the ease with which it can be done in a frozen brain, and the difficulty—or, I should rather say, the impossibility—of cutting a regular series of sections by any other method, will be convinced of this. The brain, however, must be specially prepared for freezing, as otherwise one might as well try to cut through a piece of rock, so hard does it become. Unless specially prepared, its structure will also be so damaged by the pressure of the ice crystals as to be totally useless for my method of bringing out the fibres. It must be impregnated with some mixture which will prevent it from freezing too hard. In a paper which I formerly published on the *technique* of this subject ("Journal of Anatomy and Physiology," Vol. XII.), I recommended soaking the brain tissue in simple syrup of a certain strength. I have since found out that the following is much preferable:—

	ozs.
Syrup (1 oz. by weight crystallized sugar to 1 fl. oz. of water)	4
Mucilage (1 lb. gum acacia to 80 oz. water.)	5
Water	9

Mix and saturate, by boiling, with boracic acid.

Filter through muslin.

The above strength is the only one which will serve the purpose. I have experimented with many other proportions, and have found by experience that this on freezing, after having soaked into the brain tissue, produces the best consistence for cutting.

The method of employing this freezing mixture, is firstly to get rid of the spirit by soaking the segments in water which is constantly renewed for at least three days. It is usually better to allow from four to five days, according to the thickness of the segments. They are afterwards immersed in a large quantity of the freezing fluid, so that they may be thoroughly soaked by it, each segment being separated from its neighbour as before described.

They should lie in the freezing fluid for a fortnight at least, and the longer they are immersed in it the better. I keep them permanently in this until they are wanted.

Cutting of the Sections.—The segments are now ready for cutting. The microtome which I have found to be most serviceable is one made for me by Mr. Gardiner, surgical instrument maker, Edinburgh. It is extremely simple in construction, and the freezing part of it is essentially on the same principle as that described by Professor Rutherford, there being certain necessary additions in regard to the knife. The well is nine inches in diameter, and a very powerful finely graduated screw moves a cylinder upwards on which is placed the piece of brain to be cut. The surface to which it has to be frozen is roughened by means of a series of concentric rings, so as to prevent the piece of brain from becoming detached. I fix the piece of brain on *with the freezing mixture* just described. Mucilage does not suit, as it freezes too hard and becomes brittle. The section is apt to tear in being detached in front, unless there is some mass to support it. I usually fill the well here with the freezing fluid, but at other parts I leave it unfilled, simply freezing the brain on to the top of the grooved brass cylinder.

The best freezing mixture is salt and snow, and it must be pressed in all round the well, and stirred from time to time. It freezes much more quickly and more equally if the water resulting from the melting of the snow is not allowed to run off until it becomes excessive. The quantity of snow required varies of course with the temperature, but I have found that in moderately cool weather half-a-hundred weight is more than sufficient to keep the machine frozen for a day. Ample time is thus afforded for preserving every section from a piece of brain of the thickness formerly indicated (1 inch).

The knife has given me much more trouble to adjust than the freezing part of the apparatus. The surface to be cut is so large, and the tissue when frozen so hard, that any ordinary knife constructed similarly to those employed for smaller microtomes is utterly useless. The only cutting instrument at all applicable is one made on the principle of a plane. That which I employ has a framework made of brass, and is very massive, so as to afford a considerable momentum. It is exactly like a large Canadian plane, with the front part of the framework cut out. The blade rests upon three solid metal supports behind, and is fixed to them by a screw. The blade is made of a thick plate of steel, and the whole framework runs in a grooved railway at each side of the plate of the microtome, so that lateral or other deviation is prevented. The edge of the blade being sufficiently strong not to bend, is thus bound to run through the frozen brain segment. The railway entirely does away with the necessity for having a plate-glass top to the microtome, as the blade never comes in contact with the metal in being swept forwards.

In employing this cutting machine, the railway should be well smeared with glycerine, and the blade is driven forwards with great rapidity. It must be perfectly dry, and as cold as possible. I formerly used to freeze the blade with a couple of boxes containing a freezing mixture placed in contact with it behind. The object of having it dry and cold is to prevent the section sticking to it. If this happens the section is ruined, as no amount of manipulation will be sufficient to detach it entire. It ought to be thrown off the blade, to turn once over, and to land on a flat vessel containing water about a foot and a half to two feet in front of the microtome. The beauty with which this can be done by means of this instrument requires to be seen to be appreciated. The section thus lands on the surface of the water before it has thawed, and it continues to float here usually perfectly spread out, or, if not so, capable of being unfolded by means of a couple of camel's-hair brushes. The section is so fragile and so large, that unless it were thrown off in this way I cannot see how it would be possible to prevent destroying it. Each section must be removed from the water separately as it is, cut on a glass plate prepared in the manner now to be described.

Method of Fixing the Section on a Glass Plate.—As just mentioned, the section is thrown from the blade of the cutting machine on to water contained in a flat dish in front of the microtome. Previous, however, to this, a plate of common window glass (12×11 in. is the best size), is covered with a layer of French gelatine, and placed in the flat dish. The gelatine solution for gelatinising the plate should be made in the following manner:—Soak a quantity of French gelatine (Coignet's is best) in water for half-an-hour; melt this in a water-bath, and measure the quantity of the solution. Take three times as much water, and add to it sufficient carbolic acid to make the whole mass up to a 1-to-40 mixture; gradually add the water with the dissolved carbolic acid to the warm gelatine solution. Pour it over the glass plate with a pipette, so as to make a pretty thick uniform layer.

This gelatinising of the glass plate ought to be done immediately before the section is cut. The gelatine should be allowed to firm, but not to dry, a few minutes after it is applied being sufficient for the purpose. If placed in water, numbers of air-bells will be found to adhere to the surface, but these can be easily removed by holding the plate under a gentle stream of water, and lightly rubbing the gelatinised surface with the finger. This gelatinised glass plate is to be the recipient of the section when it is thrown on the surface of the water; and as a great commotion of the water and consequent disturbance would result if it were put into water after the section had been cut, it is necessary to do so as a preliminary measure, and to allow any disturbance of the water to subside.

There should not be too much water in the flat vessel. About half-an-inch is the most that is required. If more than this is employed, there is difficulty in getting the section out on the slide. The glass plate is gradually lifted up out of the water, bearing with it the section which is floating on the surface. This slide is not to be the one on which the preparation is to be permanently mounted. Hence it is immaterial in what position it lies upon it. This is a very great advantage, as it is extremely difficult to fix it on a particular part of the plate while in its present unstable condition. The water running off from the plate tends to displace it. Whatever position it is in when removed, this should be retained, and no attempt made to alter it by means of brushes or otherwise.

The gelatinised slide, with the preparation upon it, is now placed in a gently sloping position. About half-an-inch elevation is requisite, so as to allow the water to drain away, and, at the same time, prevent the preparation sliding off the plate. This position should be maintained until the superfluous water has been entirely got rid off, but the preparation must not be allowed to dry. If any air-bells are enclosed under it, they can be drawn to the side with a damp camel's-hair brush, or dissipated by touching them with a hot needle.

The preparation at present has a layer of gelatine under it. A second layer of the same gelatine solution is now dropped over it with a fine pipette. It should be put on at a high temperature, and the aperture of the pipette should not be too large, as a gush of gelatine solution is liable to occur, which may destroy the preparation. This is allowed to cool, and now the preparation is perfectly enclosed between the two layers. These become inseparably united, and it is thus fixed upon the slide. It should be labelled at once by some distinctive symbol and number indicating its position in the series.

Method of Defining the Fibres and Enlarging the Preparation.—As looked at in this stage, one would hardly believe that the preparation is the same as it turns out to be when finished. It is perfectly opaque, and the definition of the white from the grey matter is very unsatisfactory. In order to give a demonstrative view of the fibres, it should not be extremely thin—not so thin as one usually cuts brain for ordinary microscopic work. Consequently, were the preparation retained in this state permanently, it would be of comparatively little use for the object I have in view. Some means must be adopted for clarifying it, and for bringing the bundles of fibres into prominent contrast with the grey matter. For this purpose the usual clarifying agents, such as oil of cloves, turpentine, or glycerine, are quite inapplicable. The first and second clarify too uniformly, and do not differentiate the white matter from the grey sufficiently well. The whole preparation becomes uniformly transparent. After trying many different ways of accomplishing this, I have now discovered a method which, for the purpose of demonstrating the white tracts and the grey nuclei for naked-eye purposes is, I believe, better than anything we possess. It is the following :—

The preparation, covered above and below with gelatine solution, and in its yet opaque condition, is allowed to dry. Several days' exposure is usually necessary for this end. When it dries, it becomes perfectly transparent. The gelatine prevents it from cracking or shrinking. It is converted into a thin transparent film, firmly adherent to the glass plate. The gelatine becomes thoroughly incorporated with it, so that the textures of each are practically continuous. The differentiation now is even worse than before, because the white and grey matters are equally clarified.

The dried preparation, and the gelatine for about two inches round it, are next brushed over with liquor potassæ (British Pharmacopœia). Sufficient is required to soak it thoroughly, and it should be spread over the surface with a brush, so as to equally distribute it. It is allowed to remain in contact with the preparation for five minutes, and the slide is then elevated at one end, so as to allow the superfluous potash to run off. If any gathers at the lower part of the slide, it should be brushed away.

The preparation so treated is now removed to a cool place for twenty-four hours, and if at the end of this time the gelatine be cut round at about a distance of one inch from its edge, the whole of the preparation incorporated with a film of gelatine can be stripped off from the surface of the glass. If it is not perfectly dry, it should be laid on the reverse side of the glass plate until it is so. The preparation in this state can be handled with impunity, just as a sheet of dry gelatine can. The two have become so incorporated that they appear to be one tissue. The preparations should now be labelled with black varnish, gathered into bundles, and reserved for further treatment. They keep, if retained in a dry atmosphere, apparently for any length of time, and can be sent by post, or be otherwise conveniently disposed of, as the case may be.

I discovered in working at this subject that if gelatine be dried, and treated with potash as described, it becomes so hygroscopic that when placed in water a sheet of it will expand to nearly double its original size. If glycerine is mixed with it the same swelling occurs, but to a minor extent; and I further made out that if a section of brain is enclosed, as mentioned, in the film of gelatine, it also expands to a similar degree, and, at the same time, there is produced the most beautiful differentiation of the white from the grey matter. If one of the above-described gelatine films, with the enclosed preparation, be placed in water for half-an-hour, it will be found to have grown from one half to double its original size. The grey matter remains quite transparent, while every bundle of fibres in the white matter is marked out with the utmost clearness. The beauty of the preparation in this stage is very striking, especially as contrasted with its appearance before being immersed in the water.

It can even yet be handled without much fear of injury, and can be turned round in the water, so as to get rid of any underlying air. A glass plate, similar to the one before described, is next slipped under it while in the water, and the preparation withdrawn. There is now no difficulty in adjusting it in proper position on the slide, as the casing of gelatine protects it. It might be supposed that the consistence of the gelatine would be destroyed by the action of the potash. This is not so if too much is not employed, and if it be applied only once. If there should be any difficulty in getting the preparation off the slide after the potash has been added, it is usually due to its having dried too much during the twenty-four hours which have elapsed from the time of application of the potash. If so, all that is necessary is to damp the adherent part slightly. It usually strips off without any difficulty, the potash having the effect of loosening the hold of the gelatine upon the surface of the glass.

By the method just described, a two-fold object is gained—namely, that all the parts of the preperation are much enlarged, and the white matter is perfectly differentiated from the grey. The ratio of expansion seems to be equal throughout; so that every part, although enlarged, presents the same relative size to others. The advantage of this method of enlargement is that it makes small objects more conspicuous, and at the same time, by separating the fibres, dissects them out to a certain extent, and renders the tracing of them much easier. By repeating the process, and again expanding, a still further enlargement follows; so that one can go on successively enlarging in this manner until an enormous preparation is produced. I do not know how large such preparations could be made, but I have some in my possession which are more than twice their original superficial area.

At each fresh application of potash and gelatine, the differentiation also becomes more pronounced. Nor is the preparation rendered useless for microscopic purposes. The fibres can be traced perfectly, either with a low or high power, if such should be considered necessary. By far the most instructive picture, however, is gained by unaided vision, or by the use of a simple lens of large size.

The Mounting of the Preparation.—The section having been expanded and taken out of the water on a glass plate, the superfluous water should now be allowed to run off. This takes about twelve to twenty-four hours, and it should not be further treated in any way until after this time.

It must be mounted in glycerine jelly, made in the following manner:—Soak French gelatine in water as before until it is saturated, melt it, and measure out equal quantities of German glycerine and of water, adding to them sufficient carbolic acid to form a 1-to-40 mixture. Add the water, glycerine, and carbolic acid to the gelatine in a water-bath.

A layer of this glycerine jelly must be put over the preparation and allowed to harden before the cover-glass is adjusted. The reason of this is, that in being covered it is liable to become displaced unless fixed to the surface of the glass. There are other reasons which render this procedure necessary. The glycerine jelly is dropped on the preparation from a pipette at a temperature of between 80° and 90° F. If higher than this, it will melt the gelatine around the preparation and destroy it.

A nice even surface is thus formed, over which a cover-glass can be easily slipped. If there should be any opaque parts in the preparation, it should be left in this uncovered state until they disappear. It is usually advisable to allow it to remain uncovered for three or four days under any circumstances. It will then be ready for mounting. This is accomplished by first holding it under a gentle stream of water for a minute or so, and rubbing it softly with the hand until the surface is thoroughly wet. The same glycerine jelly as before described is now placed over it, at the formerly-mentioned temperature, and the cover-glass, previously warmed, is slipped over this. In a very short time the glycerine jelly fixes, and then the preparation is safe.

The superfluous glycerine jelly outside ought during the same day to be removed, the surface washed with hot water, and the edges of the preparation cemented with the following:—

Gum Damar	8 oz.
Benzole	8 fl. oz.

Let them dissolve. Filter, and rub in with a pestle and mortar 7 drs. of oxide of zinc. Filter through muslin. The greatest trouble may be caused by air getting in at the edges, unless the cementing is done on the same day as the mounting of the preparation. The cover, it may be stated, is made of common window glass, similar to the slide on which the preparation is mounted. It should always freely overlap the edge of the preparation.

The process which I have described is specially applicable for large brain sections. It might, however, be employed for other tissues where it is desirable to keep the parts *in situ*. I should think that sections of the eye-ball would be specially demonstrative if mounted in this way.

ON LOSS OF BOTH KNEE-JERKS FROM ONE-SIDED BRAIN DISEASE.

BY STEPHEN MACKENZIE, M.D., F.R.C.P.

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I PROPOSE to discuss a very interesting phenomenon, which offers considerable difficulty of explanation, namely, the loss of both knee-jerks from a unilateral cerebral lesion. It will, I think, be found that the condition is not by any means of rare occurrence, but attention has, as far as I know, scarcely been called to it, and I am not aware of any explanation that has been advanced concerning it. It will perhaps be most convenient if I first relate a case in which the phenomenon existed. It was a case of gliomatous tumour of one hemisphere causing hemiplegia and hemianasthesia, whilst the patient had complete loss of both knee-jerks. The case is as follows:—

M. N., aged 25, came to the Ophthalmic department of the London Hospital for defect of sight, on the 8th of January, 1880. My colleague, Mr. Waren Tay, under whose care she came, noted "Myopia; changes in yellow spot region (resulting from hæmorrhage ?)

$$\text{L. } 3\tfrac{1}{2} \text{ Snellen } \frac{20}{0} - 3 \text{ D } \frac{20}{70}$$

$$\text{R. } 3\tfrac{1}{2} \text{ Snellin } - 11 \text{ D } \frac{20}{100}$$

Evidences of neuritis, with hæmorrhages, in *left eye.*" On the 15th of March, when seen again, Mr. Tay noted: "The *right* disc is now distinctly swollen, the margins obscure, and there also appear to be small nodules of lymph here and there. In addition, of course, are the myopic patches of more or less exposed sclerotic. No hæmorrhages detected in right eye. In left eye, well marked swollen disc, with lymph and hæmorrhages."

The following history of the case was elicited by Mr. Tay and Mr. Makeham, then my clinical clerk:

"Family history unimportant. The patient was backward in teething and walking, and at school, in learning. She had a discharge from her ears, accompanied by pain, from the age of three until dentition was completed. When 9 years of age had disease of right hip, following scarlet fever, which left her permanently lame. Since the age of 15 has been engaged as a milliner.

"Just before Christmas, 1879, she complained of pains in the head, and of a cold numb feeling down her right side. On the 7th of February, 1880, she appeared to lose her sight almost entirely and somewhat suddenly, but she had complained of defect of vision since the beginning of the year, and as already stated she was seen by Mr. Tay on the 24th of January, and her condition then noted. At the time when the loss of sight became nearly complete she had very severe frontal headache. One week before admission she had two fits, with an interval of fifteen minutes between them. In the fits she was unconscious, and had convulsive movements almost confined to the right side. When she came out of the fit she squinted a little, temporarily. Since the fits she has not been able to walk, has seemed very drowsy, and has slept a good deal. Mr. Tay, recognising the nature of the case, asked me to see the patient on the 20th of March, when I made the following note:

"The right hand and arm are feeble. She drags the right leg in a feeble way, and has not power to stand on it. She cannot stand or walk at all unassisted. The pain in the head is very severe, and only relieved temporarily by hypodermic injections of morphia. There are occasional movements of right eye, inwards and a little downwards, independently of the movements of the left eye. These movements are increased when she looks to the left. The external rectus of each eye appears feeble. The double optic neuritis, severe pain in head, vomiting, and slow onset of paralysis, all point to tumour of the brain."

The patient was transferred by Mr. Tay to my care on the 20th of March, when it was noted as follows: the eyeballs are prominent, the corneo-sclerotic junction being seen all round when she opens her eyes. Can just see moving objects. Tongue clean, bowels much confined. No abnormal physical signs in chest.

As already mentioned there is decided loss of power in the right arm and leg, but no facial paralysis or deflection of the tongue are detected. *There is entire absence of both knee-jerks.*

April 1st.—Yesterday afternoon facial paralysis was noticed by the sister of the ward. To-day the mouth is slightly drawn to the left. When she parts her lips the mouth is much more arched on the left side. The right eye closes imperfectly upon making an effort. The left eye winks naturally, whilst the right scarcely moves. When she frowns no wrinkles can be seen on the right side. The tongue on protrusion is slightly deflected and curved to the right. Uvula not deflected. Sensation to heat, cold, pain, and ordinary tactile impressions are impaired on the right side. She cannot move the right lower extremity. *There is complete loss of both knee-jerks.* No increase of plantar reflex on right side. She cannot walk now, even with assistance.

April 3rd.—Complains of constant headache. No vomiting. Facial paralysis still more marked. Cannot hear a watch close up to right ear. Smell absent. Taste normal.

April 6th.—Since the 2nd of April has not been able to distinguish night from day.

April 9th.—I noted the paralysis of motion of right face, arm, and leg is very complete. Owing to the patient's blindness, it is difficult to test the ocular movements, and all that can be said is that the right superior rectus does not move so freely as its fellow. There is marked, but not complete, loss of sensation on the right side of face, and the sensation of the right arm and leg is very much impaired, being only observed on deep pricking. She can now hear a watch with right ear at about one inch distant, on the left side at four inches distant. There has been no incontinence of urine or fæces.

April 12th.—Facial paralysis less marked. Can feel better the prick of a pin. She has also regained to a very slight degree the movement of both upper and lower right extremities.

April 19th.—Complains very much of pain in the head. Has vomited several times the last few days.

April 20th.—The minute vascular injection of the left disc is less conspicuous, the tissues being more opaque. The retinal veins are very large and tortuous, the arteries small, and there are a number of hæmorrhages on and around the disc. Between the disc and the yellow spot are a number of white striæ converging to the latter point. Her urine contained a trace of albumen on admission, none since.

April 23rd.—The left eye appears slightly more prominent than the right. The palpebral fissure is smaller on the right side, apparently owing to want of tone, for she can both open and close the eyelids, but she closes the right more freely than the left. The movements of the eyes are limited in all directions, especially upwards.

April 28th.—Has become more and more helpless, the pain in the head has been very severe lately.

April 29th.—Swelling of right upper lid, conjunctiva injected, and one or two abrasions over cornea. The whole of right side of face is injected, and there is a profuse mucous discharge from the right nostril.

May 2nd.—Patient complains of pain in the *left* extremities—"As if they are being pulled away from me," she says. She is losing power on the left side, this has been noticed for some days. She cannot draw up the left leg; cannot lift the left arm from the bedclothes; the grasp of the hand is very feeble. No anæsthesia on left side.

May 6th.—Suffocative attack, associated with a choking cough, in which she became livid in the face.

May 9th.—After a trifling improvement is again worse. She has had another "choking fit" to-day, this being the third. They come on after drinking something, and nurse says, "she seems as if she had not sense enough to swallow properly." Following this last attack she sank into an unconscious condition, the heart's action became irregular and intermittent, the respiration feeble and gasping, and she died at 10.10 P.M.

The *necropsy* was made on the following day. The detailed description of what was found is mislaid, but the following transcript from the abridged *post mortem* records supplies all that is essential to be conveyed:

"Glioma of left hemisphere (with hæmorrhage) involving left corpus striatum and thalamus opticus, and extending down crus cerebri to its junction with the pons. No changes in viscera except venous congestion."

The spinal cord showed no changes to the naked eye. It was hardened in bichromate of ammonia, and has since been examined. Sections from cervical, dorsal, and lumbar regions show no descending wasting, and no changes of any importance.

There are several points of interest in this case, to which I may in passing allude.

I. That the symptoms, though not strictly beginning in the eye, so far predominated there that the patient first came under the care of the ophthalmic surgeon. When first seen by Mr. Tay there was unilateral neuritis. It was the left eye which was first affected with inflammation and the disease causing it was also on the left side of the brain. As I have shown[1] when optic neuritis is unilateral it has usually been on the side opposite to the growth. Dr. Ernest Field[2] has also recorded a case illustrating this fact. But whilst I think this is often the case, it is clearly not, and I did not in drawing attention to the point intend to imply that it was, invariable. As regards the ocular movements and paresis, but little can be said, but the tumour involving the thalamus was in close propinquity to the centre discovered by Adamuk in the corpora quadrigemina and thus may be explained the movements and weakness of the muscles of the right eyeball from disease on the left side of the brain, no disease being found on the right side. Owing to the patient's state these conditions could not be very accurately studied.

II. The symptoms enabled a localisation of the disease to be made with certainty. The dysæsthesia followed by anæsthesia pointed to the thalamus opticus or its vicinity: the right sided convulsion followed by gradual and extending paralysis of motion of the right half of body, pointed, in conjunction with the anæsthesia of the same area to disease involving the corpus striatum, or anterior part of internal capsule. The severe headache, vomiting and double optic neuritis, made it certain that the local disease was tumour. The absence of syphilis, acquired or congenital, the absence of signs of decided struma, and the age of the patient rendered it probable that the tumour was, as it proved to be, gliomatous.

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[1] 'BRAIN,' vol. ii., p. 269.

[2] Ibid. vol. iv., p. 240.

III. The hæmorrhage into the tumour just before death. I regret that my detailed notes on this point are not available. There was a large black clot in the growth. We know that this not infrequently happens in vascular gliomata, that it may give rise to apoplectiform symptoms, and in the inexperienced, especially in the absence of history, lead to the growth which has bled being overlooked. This would be all the more likely when, as in this case, the growth occupied "the seat of election" of hæmorrhage.

The point to which I especially wish to draw attention in this case, however, is not any of the foregoing, but to the double loss of knee-jerk. It will be observed the cerebral disease was strictly unilateral, the loss of knee reflex double. I believe in this respect this case is by no means an isolated one. I have seen more than one case and know of others in which there has been clinical evidence of cerebral tumour with double loss of knee-jerks but in which there was no post mortem. Dr. Hughlings Jackson mentioned at a discussion at the Ophthalmological Society recently a case in which there was local brain disease, but in which no change was found in the cord after death[1] and since then I have seen the parts from a case of his in which there was unilateral tumour loss of both knee-jerks, and no naked-eye changes in the cord at the necropsy. Dr. Hughlings Jackson alluded to other cases he had seen without necropsis.

The *loss* of knee-jerks from cerebral disease is difficult of explanation. We know that the brain exerts a controlling or inhibitory power over the spinal cord, as regards its reflex functions; and that this inhibition can be increased by a voluntary effort. The exact course of the inhibitory fibres in the brain and cord is not certain, though as regards the latter it is probable that the fibres pass along the pyramidal tracts.

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[1] 'LANCET,' June 15, p. 1046.

It is well known that when the cerebral inhibition is removed, as by a destructive lesion between the medulla and the level of the cord, concerned, as a centre, in completing the reflex, exaggerated reflex is the result. How then is it to be explained that a cerebral lesion causes abolition of the reflex?

I venture on the following explanation with the greatest diffidence, and my putting it forward may have the effect of eliciting a better one. Suppose there is an inhibitory centre (or centres) in each hemisphere acting upon the spinal cord as a reflex centre. These centres under ordinary circumstances act together, and control the reflex excitability of the cord. Their inhibitory power is capable of being augmented by a voluntary effort, say by means of cortical centres connected by commissural fibres with the inhibitory centres lower down.[1] The two inhibitory centres naturally act together, each being also connected with both halves of the spinal segment concerned in the reflex, in the same way as motor impulses are conveyed to both halves of the cord by the crossed and direct pyramidal tracts. But being arranged for combined action, if one is injured or destroyed, the other is uncontrolled; and connected as it is with both halves of the cord, over-inhibits or annuls the reflex affected through both halves of the spinal segment concerned. The injured, or destroyed, normally cooperating inhibitory centre necessarily has its function abolished; and, if there were no other action, this would lead to exaggeration of the reflex governed by the inhibitory centre. But this is prevented by the uncontrolled healthy inhibitory centre whose positive action outweighs the negative action of its destroyed fellow centre. In this way can be explained how a lesion limited to one side of the brain can cause loss of both knee-jerks, and this without there being any structural alteration in the spinal cord. The diagram on page 231 will, I hope, serve to illustrate my meaning.

Let us suppose A and B, connected by the commissural fibre

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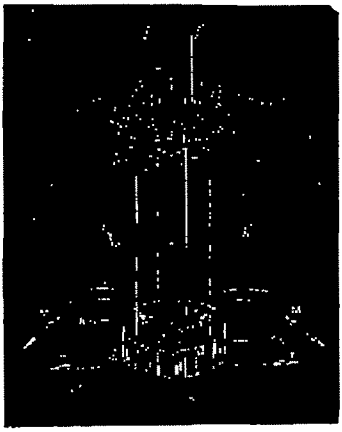
[1] This is certainly less probable as regards the knee-jerk than as regards the vesical and some other reflexes. I am aware of the powerful arguments against the knee-jerk being a true spinal reflex, but it is enough for the argument to admit, as is universally done, that it depends upon a spinal influence.

a, to be inhibitory centres in each half of the brain (R H and L H), A and B are connected with higher centres C and C′, by which their inhibitory power can be strengthened. From A fibres pass by C and C′ to the right and left of the spinal segment (S C), where, in the anterior horns (A H), they communicate with the ganglionic centres there situated, and exercise their inhibitory function. From B fibres pass by b and b' to the spinal segment in similar manner. T represents the patellar tendon (I assume, for simplicity of argument, that the knee-jerk is a tendon reflex), and M the extensor muscles moving the leg. A stimulus from T is conveyed along d by the posterior root to the spinal cord, and an efferent impulse sent by the anterior horn along e to the muscles (M). In ordinary circumstances the reflex completed in the spinal segment, say in the anterior horn, is here controlled by the fibres, say from the crossed and direct pyramidal tracts, coming from the inhibitory centre of each side of the brain. Destruction of one half of the cord above the segment concerned in the reflex, involving b and c or b' and c' will lead to exaggerated knee reflex on the injured side, by removing the influence of both cerebral inhibitory centres; and destruction of both halves of the cord above the segment will lead to exaggerated knee reflex on both sides. This is the explanation usually received.



R. H., right hemisphere of brain. L. H. left hemisphere. A. right cerebral inhibitory centre; B. left ditto, ditto. C. cortical centres. A. H. ganglion cells of anterior horn of (S. C.) spinal cord. M. muscles moving knee. T. patellar tendon. a. commissural fibres connecting cerebral inhibitory centres: b and b^1, paths of cerebral inhibitory fibres from left half of brain to both halves of cord: c and c^1, paths of cerebral inhibitory fibres from right half of brain to both halves of cord. d sensory fibres from patellar tendon to grey matter of cord: e and e^1, motor fibres from anterior horn to extensor muscles moving knee: f and f^1, fibres from cortex to inhibitory centre.

If, however, there be a lesion of one side of the brain invol-

-ving this inhibitory centre A, the influence of A is removed from both halves of the cord; but B, being unantagonised, overacts, and being connected with both halves of the cord, *annuls both knee reflexes.* A difficulty immediately suggests itself as to adequacy of this explanation. In hemiplegia, due to a unilateral cerebral lesion, there is usually exaggeration of the knee-jerk on the paralysed side. This is generally said to be caused, but more correctly described as permitted, by the secondary degeneration of the cord above the spinal segment concerned in the reflex. It would appear to me to be due to, or permitted by, involvement of parts below, A and B in *b* and *c*, or *b′* and *c′*, and if so it would not militate against my hypothesis. It will be observed in my case, that though there was right hemiplegia, there was no exaggeration of the knee-reflex on the paralysed side. These corresponded with the absence of descending sclerosis in the spinal cord, and thus harmonises with my hypothesis.[1]

As I have already said, it is not ascertained how the fibres travel to the spinal segment from A and B, or whether they are independent of the motor fibres passing from the brain to the cord. This hypothesis, it will be seen, explains how the knee-reflex is exaggerated when there is descending wasting in the cord, and at the same time explains why there need be no lesion in the cord when the reflex is abolished on both sides from a unilateral cerebral lesion, as in the case I have narrated, and as occurs probably not unfrequently. On my hypothesis, if *both* inhibitory centres were disabled, there would be exaggerated knee-reflex on both sides, from complete removal of all cerebral inhibition, in the same way that a total transverse lesion causes exaggeration of both knee-jerks. I do not know, however, of any facts bearing on this point.[2]

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[1] The increase of knee-jerk noticed after convulsive seizures would appear to be due to exhaustion of the hypothetical inhibitory centres.

[2] See a very interesting case of loss of both knee-jerks from tumour of cerebellum, in a case of Dr. Ramskill's, reported by Dr. Coxwell, 'Brit. Med. Journal,' May 19, 1883, p. 957.

ON A CASE OF "SAWYER'S CRAMP."

BY G. V. POORE, M.D., F.R.C.P.

Professor of Medical Jurisprudence, University College. Assistant Physician to University College Hospital.

THE patient whose case is recorded in the following short paper was sent to me by my friend Dr. de Watteville, who was kind enough to remember that I took an interest in "professional" ailments.

W. W., æt. 38, came as an out-patient to University College Hospital on the 8th of March, 1883. He was a maker of packing-cases and had been engaged in piece-work, making his packing-cases against time, as it were, for 10 or 11 hours a day, and occasionally for as much as 14 hours. He was a strongly-built man, but looked a little worn about the face, and had a certain earnestness of manner which I have often seen associated with genuine professional ailments. He did not drink, and there were no facts in his personal or family history which need to be recorded. His only trouble was an inability to use the saw. As soon as he took his saw in his hand, his efforts to use it were thwarted by disorderly movements which he could not explain.

In order to better understand the case, I asked him on the next occasion to bring his saw with him to the hospital, together with a piece of wood such as he habitually used in his trade. This was done, and the patient was stripped to the waist, and was made to go through the act of sawing.

On taking the saw in his hand, there was noticed a slight tremor of the blade, due to a little shake of the hand. The saw was thrust forward through the wood fairly well, but the back stroke could not be accomplished, and the attempt was accompanied by abnormal elevation of the shoulder, scapular movement, adduction of the elbow, rotation outwards of the forearm, flexion of the wrist, and swaying to and fro of the body. These movements were difficult to analyse, the general impression left upon the observer being that the whole of the muscles of the right upper limb behaved tumultuously when the patient attempted to saw.

With the left arm he could saw perfectly well.

From the character of the movements I thought it not unlikely that some of the scapular muscles were at fault, and my first step in the examination was to outline both scapulæ with a blue pencil, to see if there was any want of symmetry in the position of the scapulæ on the two sides. This examination gave a negative result.

On close inspection, there was seen to be distinct flattening of the right supra-spinous fossa as compared with the left. That this was not due to any wasting of the trapezius was shown by putting the trapezii in action, when the muscles on the two sides appeared to be equally developed. The first positive fact with regard to this patient then was—

(*a.*) Diminutions in size of the right supra-spinatus muscle.

On close inspection of the chest, there was noticed a slight degree of flattening of the right infra-clavicular region, and I came to the conclusion that there was

(*b.*) Diminution in size of the right pectoralis major, especially the clavicular portion.

The muscular wasting in the above situations, though very slight, was undoubted, and when pointed out was readily admitted by all who saw the patient.

I was almost inclined to think that there was a little want of plumpness about the right infra-spinous fossa, and that the muscular mass between the spinal border of the scapula and the spine was more flabby on the right than the left side; but of these two facts I could not feel at all sure. No other muscles except those in the situations named were in the least degree wasted.

The next step in the examination was to determine if any of the nerves of the brachial plexus were tender. There was no tenderness above the clavicle, nor over the situations of the great nerve trunks (musculo-spiral, median, or ulnar) in the upper limb.

(*c.*) There was a markedly tender spot in the right pectoral region, in the second interspace, midway between the mid-sternal line and the point of the shoulder.

(*d.*) An equally tender spot beneath the spine of the right scapula, just where the supra-scapular nerve enters the infra-spinous fossa of the scapula. The next step was to test the irritability of the muscles to faradism. It was found that the muscles of both upper limbs manifested equal degrees of irritability with one exception.

(*e.*) Both portions of the right pectoralis major (clavicular and sternal) showed a marked degree of excessive irritability, and contracted readily to a current which produced no effect on the left side. The right muscle was far more sensitive to the current, and manifested what Duchenne would have called excessive amount of electro-muscular sensibility.

It must be remembered that the position of the supra-spinatus (which was wasted) prevented its being accurately tested, since the movement of the trapezius effectually masked any movement of the muscle beneath it.

The patient stated that he could perform every movement and every act with the right hand with perfect readiness, with the exception of the act of sawing, which, unfortunately, was the one upon which his livelihood largely depended.

Upon examining the individual movements of the limb with great care, it was found that the approximation of the scapula to the mid-vertical line (by means of the rhomboids) was not so readily accomplished on the right side as on the left; and that when the latissimus dorsi was put into extreme action, he complained of pains in the pectoral region. I am inclined to think that the difficulty of the rhomboidal movement, and the pain during the action of the latissimus dorsi, were both in part attributable to the stretching of the pectoralis major which these movements caused.

Questioned as to the origin of his trouble, he stated that it began in March, 1882, and that the first symptom was a pain in the pectoral region during the back stroke of the saw (during which movement the pectoralis major was certainly put on the stretch).

It will be observed that the case lends no support to the statement which is generally found in text-books, that in professional ailments of this kind there is nothing the matter except the derangement of a special co-ordinated movement.

In this patient distinct evidence was obtained of derangement of special muscles and special nerves, but this derangement was not of a kind to attract the attention of a careless observer. On the contrary, it was only discovered after a very diligent search.

This case, again, lends no support to the theory that ailments of this kind are due to the failure of some co-ordinating centre.

Two muscles were found slightly wasted (the supra-spinatus and the pectoralis major), and the nerves supplying them were distinctly tender; at least two sharply defined tender spots were found over the anterior rosacic and the suprascapular nerves.

It will be at once conceded that an irritable condition of the pectoralis major would be likely to seriously interfere with the act of sawing.

The connection of the supra-spinatus with the act of sawing is, however, not at first apparent.

In my papers on "Writer's Cramp" I have insisted on the fact that the muscles which are subjected to prolonged strain are more likely to be affected than those in which contraction and relaxation quickly alternate, and accordingly we find that the muscles of pen-prehension more often suffer from over-use that the muscles of pen-movement.

In the act of sawing, what muscles, if any, are subjected to prolonged strain? Not the muscles of the hand, for the saw is alternately thrust and pulled, and, during the former act the grasp is released. Not the muscles which move the elbow, for in this joint flexion and extension alternate with each other during the act of sawing. Not the muscles which pull the humerus backwards and forwards (among which are the two halves of the deltoid), because when the back-pulling muscles are contracted the forward-pulling muscles must be relaxed, and *vice versâ*. Clearly the muscles which are most likely to suffer from prolonged strain in this act are those which maintain the head of the humerus securely in the glenoid cavity, for if the head of the humerus were not firmly held during the powerful swaying to and fro of the bone, the act would certainly become unsteady. For sawing, also, the humerus must be held slightly removed from the trunk. Now, the maintenance of the head of the bone in the glenoid cavity, and the slight separation of the humerus from the trunk, which are essential for the act of sawing, both demand the contraction of the supra-spinatus.

Duchenne (*Physiologie des Mouvements*, p. 73 *et seq.*), when speaking of the actions of the supra-spinatus muscles, says that it (1), moves the humerus away from the trunk and slightly forward, and at the same time rotates it inwards very slightly, and (2), it depresses the external angle of the scapula. It usually co-operates with the deltoid and serratus magnus in raising the arm, and in the absence of the former muscle the supra-spinatus and serratus magnus combined can raise the arm very considerably. He further says, "The supra-spinatus does not merely help the deltoid in its action by its slight power of raising the arm, but its assistance is needed for maintaining the head of the humerus in the glenoid cavity during the raising of the arm. I have noticed, indeed, in my experiments, that when the deltoid contracts by itself, the head of the humerus has a tendency to escape from the glenoid cavity and to be dislocated downwards." When summing up the actions of the supra-spinatus, Duchenne speaks of it as an "active ligament of the scapulo-humeral joint." It is easy to understand how necessary the supra-spinatus must be for the act of sawing, not only in holding the head of the humerus firmly in the glenoid cavity, but also in holding the humerus slightly removed from the side.

It may be noted that the clavicular portions of the pectoralis major, which was wasted in this case, and amongst the fibres of which there was a tender spot, is also indispensable for the act of sawing, not only by helping in the to-and-fro movement of the humerus, but also in holding the head of the humerus in the glenoid cavity.

In this case of "sawyer's cramp," then, there were found two muscles wasted, both of which were indispensable for sawing, and both of which were presumably subjected to *prolonged contraction* during the act.

Clinical Cases.

A CASE OF CEREBRAL TUMOUR.

BY ALEXANDER BRUCE, M.B.

THE following case is interesting from the absence of the usual symptoms of cerebral tumour, and from the simulation of hysteria, which deceived every one who saw the patient during the last four months of life, even when careful search for gross intracranial disease was made.

L. M., 45 years of age, unmarried, right-handed, formerly a nurse, was sent to me on the 21st of September, 1882, by Mr. Miller, with a history of being nervous and fanciful, and of having had a fit of some sort at each of her last three menstrual periods, and of having in July had an hysterical attack.

Previous History.—Until two years ago she had been remarkably healthy, had been of a cheerful and kindly disposition, and had always had good situations as nurse. So far as could be learned, there had been no previous predisposition to nervous disease, either in patient or in her family. Three years previously she had been present at an accident whereby several men lost their lives, and though she retained sufficient presence of mind to render valuable assistance, she seemed to have been very much affected by the circumstance, as she constantly spoke of it at home. For some time afterwards she was nurse to a very restless child, and, according to her sister's account, was very much overworked. In January, 1881, when it was proposed that she should go to the continent with the family in which she was, a medical man in the country, for a reason I did not learn till post-mortem, dissuaded her from doing so, and advised her to return home, which she did. She then came under Mr. Miller's care, and was found by him to be very nervous and excitable from no apparent cause, till she showed him a tumour in the mamma, which she had supposed was malignant. This,—a small, round circumscribed nodule, without glandular involvement—was removed in

June, 1881. The wound healed perfectly in four days. On microscopic examination the tumour was pronounced by Dr. (now Professor) Hamilton to be non-malignant. For a time after the operation she was very well. In August, her sister informed me that she began to grow very selfish, irritable, and jealous of her sisters; that she would take fits of laughing and crying (especially the latter) for no apparent reason, and that for a time she became so passionate as to be almost unbearable at home. In September, however, she was able to take a situation as a nurse, and for some nine months she seemed in perfect health. She had charge of a very delicate child, that needed constant night attendance; so that for about nine months her sleep was constantly broken. In June she began to get excitable, and to quarrel without cause with her fellow-servants; complained constantly of pains in the back, abdomen, and about one ovary, for which no local cause could be detected, and which were at the time considered hysterical. Finally her excitement became so great that she alarmed everyone by rushing about the house at night screaming, and it was only by firm moral treatment that she could be induced to exercise self-control. She was seen by two physicians, who were of opinion that her case was one of hysteria, and recommended her removal to her own home. During one of her excited turns the first "fit" occurred, and she was said to have rung the bell to summon a fellow-servant to witness it. With rest at home the excitement passed greatly away. At the next period, in August, the second fit occurred; at the third, which occurred a week before time and was excessive, a third "fit." Since this very slight ones had occurred three or four times on some days and sometimes at night. She always knocked her sister up to see those at night, and those in the daytime were stated never to occur in the presence of certain friends, and always before certain others. One occurred during my first examination of the case, and was said by patient and her sister to be quite similar to the others. She felt its onset, said, "There's one coming," grasped my hand with her right hand, shut her eyes, threw her head back, arched her back a little. There was a slight trembling of the body, but no vasomotor change nor frothing at the mouth. She answered a question I put to her, showing (what she afterwards stated positively) that there was no loss of consciousness. The whole lasted for about thirty seconds, and was followed by no headache, or any symptom except slight numbness, which passed off immediately. Our examination went on as before. The "fit" did not seem epileptiform, but such as one sees frequently in hysterical subjects.

Though the history and behaviour strongly suggested hysteria, Dr. Bennett's case warned me to search carefully for evidence of intracranial lesion. There was found absolutely no alteration of sensibility of general or special sense (smell and taste were unfortunately not examined). Vision and accommodation, good in both eyes. No hemiopia. Pupils equal, active, and of medium size. No ptosis; no strabismus. Ophthalmoscopically fundus healthy; no sign of past or present neuritis. All the reflex functions—tendon, skin, and organic—were normal. There was no headache, and patient said never had been. (Post-mortem, I learned that she had in June complained of throbbing in the head.) No scalp percussion tenderness. Intellect clear, articulation unimpaired; could converse apparently without effort. Memory good. Her manner was excitable, and facial expression strongly suggestive of the hysterical temperament. Tongue slightly furred, slight constipation. Patient stated that she had never vomited. Otherwise I detected no abnormality. There was no sign of, and no reason to suspect, syphilis.

In short, patient seemed simply excitable, and to have fits which might fairly, except for their occurrence at night, be called hysterical. There was complete absence of all the ordinary signs of gross intracranial disease. After watching the case for a month and treating with bromide of potassium and morally, and finding by the middle of October that the fits had almost entirely gone (still happening, in presence of the friends aforesaid), that she had taken regular daily exercise, was sleeping well, and eating well; and that the only symptom she showed was increasing jealousy of her younger sister (who would not allow her to keep house); and after finding on a second examination the same complete absence of symptoms, I felt justified in excluding gross cerebral disease. In the hope of enabling or inducing her to control her temper, and as she was proving utterly unmanageable at home, she was recommended to go to the country for a time. She left town in the beginning of November; wrote after ten days that she was very well, etc. (The letters, which I saw post-mortem, were clearly written and well expressed; and I also learned post-mortem, from the lady in whose house she was, that nothing particular was seen wrong with her). On Nov. 17, she was brought into town; she had had a "fit" (the only one she had had); was said to have "paralysis"; a medical man had seen her, and had recommended treatment by "firmness with kindness." I learned post-mortem that she had summoned a servant to see the fit, and who, when it was over, said, "If that's all your fit, you needn't have troubled me." On Nov. 18 she was quite well in the morning; knitted some reins to send out to the children, without showing any sign of awkwardness in the use of her hands; went out shopping for four hours. In the evening she began to "talk nonsense," became unintelligible, and was supposed to be going insane.

November 19*th*.—I saw her in the evening. She was in bed. Mouth twisted slightly to the *left* side, *left* occiputo-frontalis weaker than *right*, slight convergent strabismus, slight weakness of right hand. Legs equally strong. No ptosis: pupils equal, active, of medium size. No optic neuritis. No alteration of sensibility, general or special. Speech peculiarly altered. No ataxic aphasia. Some, especially leading, questions were answered at once, and correctly; others after a pause, and apparently after trying to read in my face what she ought to answer. When she failed in this, she answered as if shamming stupidity. Occasionally she put her hand to her head, and said, "Oh, dear me!" as if annoyed at her loss of memory.

After my visit she got up, walked about for an hour, arranged the bed herself, using her right hand without sign of weakness.

Next day Dr. Brakenridge saw her with me. We found no strabismus, *no twist of the mouth, no inequality in the two occipito-frontales*, or in the *strength of the two hands or legs*. The grasp of the hands was fairly strong. The tendon and skin reflexes were perhaps slightly exaggerated, but equal on both sides. So far as we could judge, sensibility to touch, pain, temperature, on either side was unaltered. Watch heard with right ear at one yard, with left at one yard and a half. The speech had still the same peculiar character, not easily described, but which suggested to each of us that she could answer when she liked, and when she didn't know what was the proper answer to give she "hedged," or shammed to be stupid; thus she answered leading questions at once, and when asked on which leg she felt the prick of a pin best, she said, "It's not so well on that leg, as it's worse on the other." Unfortunately we do not remember any of her other remarks, but they were somewhat similar. She seemed in all cases to understand our questions.

Her whole behaviour, the anomalous nature of the facial pareses, their disappearance, and that of the weakness of the right hand, and the peculiar speech, suggesting irresistibly that she was shamming, and the absence of all signs of gross intracranial disease, seemed to point to a functional cause. A somewhat guarded prognosis was given, and her removal to the Royal Infirmary was recommended.

On the following day she seemed quite well, talked quite rationally during my visit, said there was nothing wrong with her, that she could be treated at home quite well, and finally began to cry, or rather seemed as if pretending to cry, as if she thought it necessary to make a strong objection to leave home. Her sister told me that after my two previous visits she talked quite well for a time, and then relapsed again; that on the previous afternoon she had had a struggle with her, and that my patient had nearly overmastered her, and that she didn't believe in her weakness.

I saw the patient only once after this—three days before death. She was sitting by her bed-side, with a stolid expression; otherwise she looked well. On my asking, "How are you to-day?" she answered with a rude stare, "Have you been sleeping?" and similarly to one or two other questions. I could not say whether she understood the questions, or whether she took this means of preventing any conversation. Some time afterward she looked as if she had discovered that she had made some mistake (but this may be an error on my part). She could walk quite well.

Dr. Mackie Whyte, resident physician, communicated the following facts. For the first few days she seemed quite well, only hysterical, wouldn't do what she was told, could only be induced to do light work, such as dusting the tables, and when the nurse was by her side, and did so then in a careless, indifferent manner, desisting the moment her back was turned. One day she vomited, and spat into her soup, which she didn't like; was supposed to have done this in order not to have to take it. When scolded for this, did not repeat the offence, except once, on the Saturday before death, when she spat into her tea. On admission: talked quite correctly to Dr. Whyte and the staff nurse: would not do so to a probationer; after a few days, began to be confused at the end of a sentence, would transpose words in a sentence, saying, "Fine—the day," for "It is a fine day." Two days before death she frequently used wrong words—e.g. "jujube" for "hospital" and "a pair of legs" for "two pills." On the day before death her father did not observe anything remarkable in her speech. Once or twice she answered Dr. Whyte as she had replied to me, but on being checked for it gave correct replies. When asked to write what she wished, her writing was unintelligible. She seems generally to have understood what was said to her, and carried out complicated directions correctly. Two days before death, after being warned by the other patients that the nurse would be angry because she spat into her tea and poured it on the floor, she went for a cloth and wiped it up. The day before death, at the morning visit, when told by Dr. Whyte to arrange her hair, which she had left hanging about her shoulders, she did so at once. Two days before death she dragged sometimes one and sometimes the other foot slightly, and vomited once (next day, the 3rd of December) quite suddenly, when asked to walk round a table. During the night she did not sleep, but got out of bed often; folded and unfolded her clothes.

December 4th.—On getting up in the morning, she slipped to the ground, became unconscious, and, on being put to bed, remained as if sleeping quietly for four hours. Half-an-hour before death, breathing became stertorous, and the pulse could not be counted. There were no convulsions.

Autopsy.—Extract from Dr. Byrom Bramwell's report: "Skull cap natural, dura somewhat adherent to skull at vertex. Convolutions markedly flattened, sulci effaced. Surface of brain sticky, but no lymph. Left hemisphere distinctly larger than the right, the enlargement being in great part due to the swelling of the left temporo-sphenoidal lobe. Weight of brain, 47 ounces. Optic discs natural. On microscopic examination, optic discs perfectly healthy." The brain was handed over to me, hardened for three weeks in saturated solution of zinc chloride, and vertical transverse sections were made at the level of the lines in Fig. 4. A tumour was found extending from the lip of the left temporo-sphenoidal lobe to line 4 (which has just missed it), at the junction of the anterior with the middle third of the pons. The dark line in Figs. 1, 2, and 3 indicates the outline of the tumour, but gives an idea of too sharp circumscription. A naked-eye examination seemed to show that it had grown from the darker part, and to have infiltrated round about, the whitish lines representing probably the remains of nerve tissue. In Fig. 2, below the tumour, there is a hæmorrhage of about the size of a hazlenut. The surroundings were pushed aside, the basal ganglia with the internal capsule being driven inward, the insula upwards, and the convolutions of the temporo-sphenoidal lobe outwards. The claustum, except perhaps in Fig. 1, was indistinguishable (partly, probably, owing to the method of hardening). The cortex was uninvolved, there being always a depth, after hardening, of $\frac{1}{8}$ to $\frac{1}{4}$ inch between the tumour and the surface. The condition of the first temporo-sphenoidal convolution is most interesting, in connection with the interpretation of the speech peculiarity. In Fig. 1 the white matter is slightly infiltrated; in Fig. 2 rather more extensively, though not so much as in the diagram. In Fig. 3 it had completely escaped, and there was a deep fissure between the tumour and the centrum ovale there. Coronal sections at the level of the tip of the lobe showed that a small part of the tumour had diverged and spread forwards into the island, about half an inch below the surface. Throughout it was impossible to distinguish the growth from the external segment of the lenticular

To illustrate Dr. Bruce's Case

Fig 1. Section at level of line 1

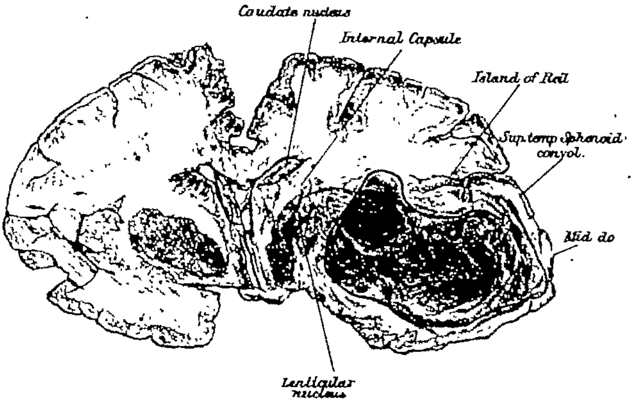


Fig. 2. Section at level of line 2

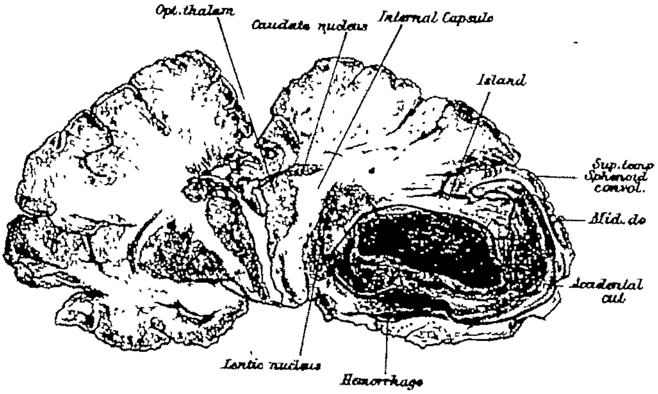


Fig. 3 Section at level of line 3

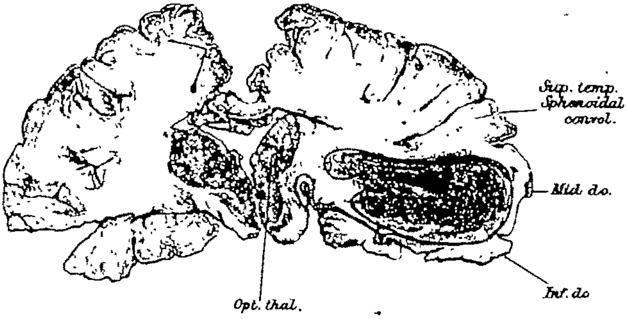
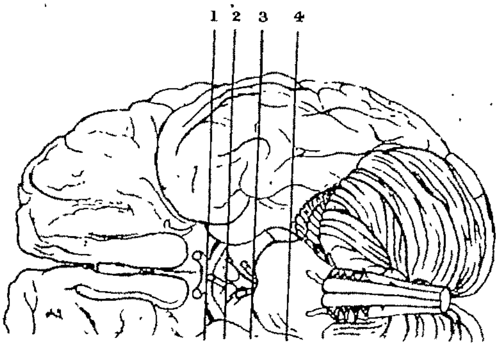


Fig 4. To show lines of Section



W & A K Johnston, Edinburgh

nucleus. Judging from a macroscopic examination (owing to the mode of hardening—the only one possible), the starting-point may have been between lines 2 and 3, and the growth from there forwards, backwards, and downwards. Pons, medulla, and cerebellum were much altered in shape by external pressure.

The following facts were learned post-mortem. Dr. Gordon of East Linton, whom she consulted in January, 1881, then found right facial paralysis and anæsthesia, some excitability of manner, and confusion as to names; also deafness in right ear (supposed to be catarrhal), complaint of a confined feeling in the head, sometimes amounting to actual pain, and a history of having on one occasion lost her way near home in a well-known street, and of having to be led home by a policeman. He supposed then that there was serious intracranial disease, but could form no idea of its nature or seat, and he was much astonished to hear that she had recovered sufficiently to again act as a nurse.

Mrs. B.—her mistress during 1881–82—found her a very good nurse, devoted to the child; but easily tired, and then occasionally dragging one or other foot. Sometimes complained of throbbing in the head; finally became excited towards night, and alarmed Mrs. B., who had been recently confined, by shouting through the house at night, and endeavouring to get into her bedroom, which had to be kept locked. During the last month she had frequently complained of nausea, and on several occasions vomited. She was supposed to be very hysterical, and finally to be going insane.

The letter, part of which was lithographed, was written on the day in which she returned from the country.

CASE OF EPILEPTIFORM MIGRAINE.

BY T. CLIFFORD ALLBUTT, M.D., F.R.S.,

Senior Physician to the Leeds Infirmary, &c.

C—— K——, æt. 24, a timekeeper, admitted May 23rd, 1882. His mother, before her death at the age of 62, and one sister who is still living, were subject to occasional attacks of dizziness; and on one occasion he can remember that a brother, without any assignable cause, momentarily lost control over his speech. With these exceptions, there is no family history of nervous affection in any shape or form: no idiocy; no epileptic or other kind of fit; no chorea.

When about eleven months old had three fits in one day, brought on, it is said, by exposure to the sun; but beyond the ordinary diseases of infancy and childhood, he has had no severe illness of any description.

There is no history of syphilitic taint, either congenital or acquired; he has never had any serious blow on the head nor fall; is quite temperate and regular in his habits.

For the last two or three years has had a good deal of mental excitement and worry.

At the age of 18 he went to sea for a year, and on his return went into the building trade, though not as a labourer; for the last eighteen months has been at his present occupation in various factories, the work requiring a good deal of physical exercise and mental activity. He distinctly remembers that when quite a boy he was subject to attacks of dizziness, which have continued up to the present time, and the attendant phenomena to be described below have either developed, or, as is more probable, have been more carefully observed. At any rate, he himself associates the dizziness with his present condition.

The attacks have become more regular and more intense during the last two or three years, and may be said to have culminated in one which occurred in the streets of Selby, about seven or eight months ago, and in which all the subjective symptoms, to be presently described, appeared.

Since this attack in Selby he fancies that his memory has failed him to a considerable extent, so much so as to oblige him to give up his work.

The seizures are described by the patient in the following manner:

"First there is a semiflexion of the fingers of both hands, and numbness of the feet, which semiflexion and numbness are relieved by the local application of cold, and increased by heat.

"Then the tongue feels numbed and too large for his mouth, and coincident with this tongue trouble is a loss of power of speech; he feels that he knows exactly what he wants to say, but on attempting to say it, probably finds that he is using altogether wrong words, though his articulation of the words themselves is perfect. In the same way with writing; he takes up the pen with the confidence of being able to use it, but finds instead that he has made a series of meaningless scratches on the paper.

"Next his sight gets dim; he can perceive light, but all objects are blurred or only half seen, and the letters of a book seem misplaced. The pain in the tender spot in the left temple, which has continued throughout, and which frequently precedes the attack by twelve or sixteen hours, now becomes much worse, and is often associated with a pain, though slighter, in a corresponding point on the right side.

During the whole of the attack he feels sick and tries to vomit; for he finds that if in the course of a couple of hours or so he can succeed, that there is a very rapid abatement of the symptoms; deep sleep intervenes, and he wakes up in the course of an hour or two, somewhat languid and tired, but otherwise quite well; better, in fact, than he was during the few hours that immediately preceded the seizure.

"If, however, he does not vomit, the symptoms become aggravated; the hands clench tightly; the toes curl downward; dyspnœa, and a feeling of faintness and oppression about the chest, come on, and it is then some eight or ten hours before the attack wears off, the subsidence of the symptoms being associated with drowsiness and deep sleep, as in the former case.

"Consciousness becomes indistinct; it is never completely abolished, and he is clear enough in his own thoughts, though he does not quite realise what is said about him. He retains, even in the most severe attacks, sufficient command to prevent himself from falling, though the spasm of his toes and legs often obliges him to sit or lie down."

When a boy, the intervals between the attacks of dizziness varied considerably; sometimes he would have as many as six or seven in one day, at others only one or two in a week or more.

During the time he was at boarding-schoool (between the age of 10 and 14 years) he does not remember having a single attack, though they invariably recurred on going home for the holidays.

While at sea, and for eighteen months after his return, there was almost a complete immunity from the seizures; and as a general rule he has noticed that they are more frequent and more severe when he is out of work, than when he is busily employed. For the last seven months, up to within three weeks before admission, the attacks have been weekly.

He is particularly liable to a seizure on the day after partaking of a heavy meal, especially if the meal contains anything "rich."

He is a healthy, well-nourished looking man. He describes himself as being very nervous and short-tempered, though during his stay in the hospital he was particularly cheerful and obliging. He seems to prefer to lie upon his right side in bed, though for no definite reason. He suffers slightly from palpitation, and says that on rising from the stooping position he has to move slowly, on account of a sharp pain, which otherwise catches him in the left side immediately below the ribs. Tongue moist, covered with thin white fur. No abnormal physical signs nor evidence of organic disease.

Tactile sensation over the body and limbs appears to be perfect, but over the forehead there seems to be greater sensitiveness on the left side than on the right.

At a point about 1 inch above the external auditory meatus of the left side, and about ¼ inch anteriorly, is a very tender spot, the size of half-a-crown, the pain and tenderness of which is much increased during the attacks, though externally there is nothing to indicate any difference between this spot and the surrounding skin. A similar point, the size of a pea, can be made out on the corresponding right side, 2⅜ inches from external auditory meatus, 3 inches from outer corner of orbit, though this is neither so tender, nor so subject to pain as the spot on the left.

Cannot now drink alcohol in any form without undue excitement, though this was not always the case.

Remarks.—The case here recorded lies upon the confines both of migraine and epilepsy, and illustrates that relation of migraine to other nerve-storms which has been so clearly shown by many modern physicians, and by Dr. Edward Liveing in particular. The attributions by K—— of many of his attacks to "rich food," on the one hand, and to "worry and overwork" on the other, is notable, as is also the banishment of the malady during the months of sea life. The very interesting connections of the symptoms with Dr. Ferrier's centres is sufficiently clear to any who will read the case with a map of such centres before him, nor will the reader forget how very commonly migraine is associated with aphasia.

A man of some 35 years of age consulted Dr. Clifford Allbutt a few years ago for migraine, the left-sided headache, when more intense, being associated with almost complete aphasia, and with transient enfeeblement of the right hand and fore-arm; and in many cases of migraine a degree of aphasia may be observed. At the same time it must be remembered that, localization apart, general brain fatigue would probably make itself felt in the order of its higher endowments and more complex work.

Speech, being the highest psychomotor function, would thus first feel the failure of general brain-power, and dexterity would be the next in order to suffer. For instance, an English Alpine climber towards the end of an arduous excursion will find the language he speaks worst to vanish from him. If this be German, he may still retain some conversational power in French; but finally this will soon also leave him, and he will be reduced to his mother-tongue. Even this fled from Professor Tyndall on one occasion when, after a grievous climb, he found on entering the Grimsel that he was incapable of articulate speech. Still, when due weight has been given to increasing exhaustion, it seems probable—from the convulsive movements in C. K.'s case, from the visual phenomena of many cases and other special symptoms observed in not a few, that migraine has some partial seat or passage in the brain, and is not altogether a superficial neuralgia.

Finally, however strong the evidence that migraine is a primary neurosis, and however often its recurrences may seem to be due to causes acting directly upon the nervous system, yet it is impossible to ignore the testimony of many sufferers who attribute its recurrence at other times to "bilious" dyspepsia. Moreover, the appearance of migraine in gouty families must not be forgotten, nor the curious fact that the migraines of gouty families are often, to all appearance, the most purely neurotic in character.

TWO CASES OF TABES DORSALIS WITH JOINT-DISEASE.

BY J. A. ORMEROD, M.D. (OXON), M.R.C.P.

THE subjoined cases illustrate the two varieties of this disease, which Charcot describes as "malignant" and "benign." In the first, the disease advanced to destruction of the joint, producing a kind of dislocation; in the second, the joint recovered so far as to be practically, though not perhaps anatomically, sound, and that too while the other symptoms of the disease were progressing.

CASE I.—Vomiting; pains between shoulders and legs; unsteadiness of gait; sudden fall, followed by swelling of thigh; disease of hip-joint.

Wm. B., 58, ship-joiner, came to the out-patient room at Queen's Square on Aug. 16, 1882; his chief complaint being lameness of the left leg; he walked with support, the left leg seeming to give under him. He said that nine months previously, while walking, a sudden pain caught him in the left hip-joint, and caused him to fall; the thigh swelled up, and he had been lame ever since. Examination showed that there was some affection of the left hip-joint. From the small and inactive character of the pupils, and the absence of the patellar-tendon reflex, it was surmised that the case was one of locomotor ataxy, and he was subsequently admitted into the hospital, where, by the kind permission of Dr. Buzzard, he remained under my care.

The account taken shortly after admission (October), for much of which I am indebted to Dr. Coxwell, is as follows:

History.—He considers that his health was pretty good till two years ago. Ten years ago he had gout in the left foot. He has had gonorrhœa and a bubo, but not syphilis, to his knowledge.

Five years ago he began to have attacks of vomiting; these used to last a day or two, or sometimes a week, with intervals, roughly speaking, of a month. The vomiting might occur before, after, or independently of food; watery stuff was brought up. It was not preceded by epigastric pain. The last attack occurred two years ago.

Pains between the shoulders, like a knife or pins running into him, began three or four years ago: he has had pains in the legs for two years. He says he has a "nervousness" in his legs, and sometimes explains this to mean that a sudden pain catches him while walking, and makes him unsteady.

In November 1881, while walking, the nervousness caught him and he fell; he got up and walked home (a quarter of a mile), and even went out again; but he again fell, on account of the pain, was carried home and went to bed. He could move the leg in bed, though not so well as now. It began to swell (he thinks in two or three days' time) first in the upper and outer part of the thigh, then in the groin. There was (he says) no redness or tenderness, nor any pain except the "nervousness." It appears that there was some inco-ordination before the hip became affected; for he states that for two or three months previously he would hesitate on coming to a crossing, and felt that he could not walk in the dark.

Present condition.—He is gray and thin; looking older than 58. Very deaf; somewhat apathetic in his ways. Pulse 90; arteries hard and tortuous: a systolic murmur at the heart's apex, and an accentuated second sound over the aorta. No albuminaria. He totters when his eyes are shut, or when his feet are put together, Vision of right eye good (excepting slight hypermetropia): optic disc and fundus normal. There is an opacity of the left cornea. Pupils 2 mm. in diameter, no contraction to light, and very little during accommodation. No squint. After removal of a plug of wax from either ear, the hearing distance was, for right ear—contact; for left, 8 inches (watch of 4 feet or more): tuning-fork on forehead or nose best heard with right ear. Both tympanic membranes are opaque and thickened; the right so much that the outline of the malleus cannot be traced. There is some numbness of the feet, but no actual impairment of sensation, either of touch or pain. The cutaneous reflexes are present (the right plantar better marked than the left): the patellar tendon reflexes are absent on both sides.

In Scarpa's triangle (left) there is a swelling, roughly oval, somewhat doughy, over which passes the femoral artery. The great trochanter is thickened, and lies nearly an inch above Nélaton's line. The thigh is movable upon the pelvis in every direction, and a grating can be felt as it is rotated outwards. There is no pain. The position of the limb (as the patient lies with his legs extended) is very much that of the other limb; and can be varied by him: there is over an inch of shortening. These conditions appear to indicate that there has been some destruction of tissue at the head of the femur, producing displacement upwards, if not actual dislocation, of the shaft.

The muscles of the left thigh look smaller than the right (girth at corresponding points R. = 13 inches: L. = 12½): but the thigh-muscles on both sides contract under the faradic current. There is some thickening about the metatarso-phalangeal joint of the right great toe: the gout, according to the patient, was in the other foot.

Since admission there has been slight subsidence of the tumour in the groin, and he has become rather firmer on his legs: he has had little pain or trouble of any kind.

Case II.—Girdle pain: affection of toe: paræsthesiæ and inco-ordination of legs: vomiting, diarrhœa: affection of knee-joint: optic atrophy: numbness and inco-ordination of hands.

[Notes of the earlier condition of this patient were published by me in the Reports of St. Bartholomew's Hospital, vol. xvii. p. 42. His condition has materially altered since that time.]

Henry S., 47, mason; out-patient at Queen's Square Hospital. When first seen (August 3, 1881) there was well-marked inco-ordination of the legs, anæsthesia of the greater part of the feet: absence of patellar tendon reflexes: there was a cataract in the right eye; the pupils were small, unequal, contracting neither to light, nor during accommodation.

He had had tightness round the abdomen for eighteen months; shooting-pains (right leg and groin) only a few months.

He had been subject for four or five months to attacks of vomiting, lasting two or three days, recurring about once a fortnight. This vomiting recurred twice while under treatment (August and October, 1881), being accompanied on the first occasion by diarrhœa after food, and throbbing sensation at the rectum, with some kind of discharge.

There was said to be no joint-disease: but it was noticed that in the left great toe the bone of the first phalanx and the distal extremity of the metatarsal bone were thickened. He said that (in 1880?) he one day took off his boot, and found the whole of the toe swollen and black; the nail came off in a week or so. There was no pain, and nothing to account for it. He has never had gout, but had what he calls slight rheumatism of that foot some years ago.

There was no direct history of syphilis: he had one child living, about eight years old, three born dead since that time.

On October 19, 1881, he came complaining of his right knee-joint. Ten days previously, on rising in the morning, he noticed a large painless swelling there. The joint is now full of fluid, bulging on either side of the patella, and for some inches above it. The swelling does not extend into the muscles of the thigh or leg; the outline of the distended synovial membrane is clearly mapped out. There is no redness, heat or tenderness. There is no crepitation or grating felt on manipulation, but he says himself that it "cracks." The account he gives of it is this: a week before the swelling came on he got out of bed in the night, and jammed his knee against the bedstead: he had some difficulty in getting it free. A few days after, while walking in the street, the knee "slipped." Nothing further followed till he found the knee swollen in the morning. There was no pain.

The subsequent course of this joint-affection has been as follows:—During the next fortnight the swelling went down a little, becoming less tense: on November 1st, slight swelling was apparent above the limit of the synovial membrane. During December the knee became more swollen than ever, measuring 15 inches as against 12¾ on the left side. During January, February, and March, 1882, the knee-joint fluctuated in size, subsiding on the whole; but there was well-marked effusion above it, beneath the muscles of the thigh. By July 18th, the effusion both in the knee-joint and in the thigh had subsided, and the condition now (November 1882), which promises to be permanent, is this: largest girth of right knee, 13 inches; of left, 12½: in the right knee some thickening both of the bones and of the synovial membrane, but movements natural; a shower of crepitation to be felt when he flexes and extends it. No pain. The muscles of the left calf and thigh look smaller than those of the right; on both sides, however, they act to the faradic current. There is a patch of superficial ulceration at the inner and lower part of the left thigh, produced, he says, by striking the other thigh against it. I may add that in the left great toe-joint (previously alluded to) there is more lateral mobility than in the right, and the nail is thick and horny.

In other respects, the nervous disease has progressed since the patient first came under observation. Thus in November 1881, a "mistiness" of the left (previously sound) eye was complained of, and the vision has since failed gradually. Now there is well-marked optic atrophy; colour of disc, grey-yellow; edges well-defined; arteries perhaps rather small; vision almost nil. The vomiting and diarrhœa seem to have stopped permanently.

The inco-ordination of the legs has become extreme, and is spreading to his arms. In November 1881, shooting-pains in the arms were complained of; in March 1882, numbness of the fingers; now, when he puts food to his mouth, it often goes to his nose.

His wife says he has had a kind of fit, in which he seemed to lose consciousness and laughed. This once happened before; and he used formerly to talk strangely.

As a resemblance (perhaps superficial) to the other case, I may mention that he is slightly deaf with the right ear. The membrana tympani is thickened and irregular, showing a small perforation in its centre. He had formerly discharge from this ear, and has complained throughout of noises in the head.

This affection of the joints appears to be distinguished by the clinical, rather than by the anatomical, characteristics, and notably by the manner of its onset. The points laid down by Charcot in his lectures are—that it occurs at an early stage of the nervous disease, before inco-ordination has set in; that it arises without apparent cause, such as a blow or a fall, and without premonitory symptoms, except perhaps crackling; that although the onset is acute, it is unaccompanied by pain or sign of inflammation; that over and above the swelling of the joint, there is swelling of the limb, partly due to effusion, partly to solid induration.

The two important features—painlessness and œdema of the limb—are sufficient, I think, to characterise the joint-affection, in the cases just detailed, as different from an ordinary acute synovitis. As regards the stage of the spinal affection, well-marked ataxy was observed in Case II. before the knee was attacked (the toe may possibly have been affected before ataxy set in); and in Case I. the history makes it probable that the patient was ataxic before the hip-disease began. A possible connection with traumatism cannot be eliminated in either case; in Case II. either a blow or a slip; in Case I. a fall, probably occasioned by the sudden giving of the legs, which so often occurs in ataxic patients. Debove has a theory[1] that in these cases the head of the bone is diseased, so that slight strains are sufficient to tear off minute pieces of bone from the point of insertion of the capsule, in this way causing synovitis.

Lastly, the coincidence of joint-disease and of gastric crises, upon which Dr. Buzzard has insisted,[2] finds an illustration in these two cases.

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[1] 'Archives de Neurologie,' July 1881.

[2] 'Clinical Lectures on Diseases of the Nervous System,' pp. 251 foll.

TWO CASES OF MÉNIÈRE'S DISEASE.

BY ALEXANDER M. M^cALDOWIE, M.D.

CASE I.—On the 10th of December, 1880, I was sent for hurriedly to see Mrs. H—. aged 43, who had fallen down a flight of stairs. I found the patient in bed vomiting severely. She complained of giddiness, nausea, and a loud singing noise in the left ear. There were several bruises on the arms and side but none on the head. The pupils were natural and reacted readily to the stimulus of light; the intellect was clear. Any attempt to raise the head brought on a severe attack of vertigo, the patient experiencing a sensation as if she were falling backwards. Each of these attacks ended in a fit of vomiting. On examination a watch could only be heard one inch from the left ear, the hearing in the right ear being normal. There was a slight purulent discharge from the left auditory meatus.

The patient stated that she had suffered from deafness and noise in the ear for more than six months. She describes the noise as being of a singing or buzzing character. She was subject to fits of giddiness accompanied with nausea and vomiting. Just before and during those exacerbations of her complaint the noise became very much increased. She had never fallen before during a fit. She stated that she did not trip her foot at the top of the stairs but fell down from giddiness.

I ordered a saline purgative, and a blister to be applied to the back of the ear.

The following day the noise remained as before, although the giddiness and nausea were less severe. There had been no vomiting since two o'clock A.M.

On the third day the patient returned to her home in Liverpool.

CASE II.—Sarah H—, aged 31, married, came to me in August last complaining of attacks of giddiness and sickness. About two years before the patient had a severe fall, injuring her right side, but she stated that her head did not sustain any injury. She suffered a good deal from shock after the fall. Ever since, she had been troubled with vertigo and a constant ringing noise in the right ear. She suffered much, at irregular intervals, from attacks of "swimming in the head," when her body seemed to rotate round a vertical axis. She could not state whether the rotation took place from right to left or vice versa. Each crisis was heralded by a shrill whistling noise in the right ear. These seizures almost invariably terminated in a fit of vomiting.

She could hear a watch only when in contact with the right ear, the membrana tympani was thickened and opaque. The left ear was normal. The heart was dilated, the first sound prolonged and harsh; the pulse 124, weak. Tongue clean, appetite good, bowels regular.

I ordered iodide of potassium to be taken internally, with counter-irritation over the right mastoid region. The patient gradually improved, the "fits" became milder and less frequent, and in about six weeks she left off treatment, being nearly well.

Remarks.—The above cases of Ménière's disease are interesting, specially from a clinical point of view. They are not examples of the grave form of this malady, described by some French authors, yet the symptoms of disease of the semi-circular canals are well marked in both cases. This disease in its less severe form is apt to be overlooked in general practice and the case treated as one of gastric vertigo or epilepsy. Case II. had consulted several medical men in this district and had invariably been informed that she was merely suffering from indigestion. She had undergone several courses of treatment but without deriving any benefit. This case illustrates the value of counter-irritation strongly recommended by Professor Charcot and other writers. Charcot consides quinine almost a specific for this disease. I regret that I had not read his lectures at that time and therefore did not test its value in this case.

In Case I. the patient maintained that the symptoms were the result of injury to the head produced by the fall, and it was only by persistent questioning that I elicited the fact that the hearing was impaired on the left side, and that she was subject to attacks of vertigo.

It is interesting to note that in one case the patient invariably experienced the sensation of falling backwards, whilst the other patient always felt as if her body were rotating on its vertical axis. Professor Crum Brown states that one canal can be affected by and transmit the sensation of rotation about one axis in one direction only, and that each pair is sensitive to any rotation about a line at right angles to its plane or planes, the one canal being influenced by rotation in the one direction, the other by rotation in the opposite direction. Notwithstanding this the cases narrated above do not appear to indicate disease limited to one canal. For cases are on record where the motor hallucinations changed their character from time to time; the rotation at one period being always apparently forwards, whilst at other times it would either seem to take place in the contrary direction, or round a vertical axis.

Reviews and Notices of Books.

Das Gehörorgan der Wirbelthiere—Morphologische histologische Studien. Von GUSTAF RETZIUS, Professor, &c., in Stockholm. Folio, Stockholm. Feb. 1881. 1. *Das Gehörorgan Fische und Amphibien* (with 35 plates).

WE regret having been so long giving a notice of this important work which may be regarded as a masterpiece in its department of Comparative Anatomy. It is dedicated to the memory of the author's illustrious father, Andreas Retzius, and certainly no more appropriate tribute could be offered by his son to the untiring genius for research of that most able and amiable anatomist.

This part forms the first instalment of a complete work on the organ of hearing in vertebrate animals which its author had planned as early as 1871, but which at one time he had almost been inclined to abandon after the publication of several of the admirable memoirs of Hasse on the same subject. But fortunately for science he was led to continue his observations and to bring them together for publication; and it may be confidently hoped that the second part of the work may, according to the author's expectation, ere long appear. The part at present under review comprehends fishes and amphibia, and the remaining part will embrace reptiles, birds, and mammals.

The work presents all the characters of having been executed with great care, both as regards the 220 pages of descriptive text, and the 35 beautiful plates, containing in each on an average between 13 and 14 figures, and in the careful reference letters and description. In fact it may be looked upon as a model for such works both by the clear description of the large amount of original works which it affords, and by the copious reference to the other researches the publication of which has preceded that of the author.

Through the kindness of a number of his friends and correspondents and his own opportunities, the author was enabled to include in his investigation and description as many as 48 fishes and Dipnoi and 15 Amphibia; and he promises the description in the succeeding volume of 22 reptiles, 10 birds, and several mammals.

The following is an abstract of the principal statements made by the author in the first part of his treatise:

In the Cyclostomata the structure of the organ of hearing stands so much below that of other fishes that it scarcely admits of comparison with them. In Myxine there is a saccus communis with one semi-circular canal (external) and two ampullæ; but while the saccus may be compared to the sacculus of other animals, the single canal may perhaps rather represent the utriculus.

In Petromyzon (fluviatilis) there are two semi-circular canals corresponding to the anterior and posterior of higher animals, with two ampullæ presenting a peculiar trifid subdivision, the middle part of which in each corresponds to the true ampulla and presents the crista acustica. The commissure of the two canals is probably the same as in other animals, but the nature of the vestibule is somewhat doubtful. There is probably an aqueduct of the vestibule or endolymphatic duct, and it may be that the elongated macula acustica combines those of the recessus utriculi, the sacculus and the lagena cochleæ; but as these are not separate some doubts may exist regarding them, and there is thus a considerable gap between the structure of the hearing organ in Cyclostomata and that of other fishes.

Among the remaining fishes, various as is their structure in many other respects, it is surprising what general similarity prevails in the structure of the hearing organ.

Among the Ganoids, Acipenser is the forerunner of the Osseous ganoids (Lepidosteus) on the one hand, and of the Teleoster on the other, but in the structure of the labyrinth it comes very near to the Teleostei. A large canalis utriculo-saccularis and a ductus endolymphaticus are present. The otolites have not yet acquired the hardness which they possess in Teleostei.

In the Osteoganoids there are true hard otolites. The lagena is only slightly separated from the sacculus. The endolymphatic duct is wide and ends as a cæcum superiorly.

The variations in the form of the hearing organ in Teleostei are so great that it is difficult at first to reduce them to one type, but a closer examination shows them to be essentially similar. Hasse has distinguished two types among them, viz. 1, that of the Perch, and 2, that of the Cyprinoids; and there is perhaps a third also, viz. that of the Clupeidæ.

Great variation in the size of the sacculus is to be found: it being very large in Gobius and Trachinus, very small in Leus, and reduced to a minimum in Exocetus. The lagena also is very variable; in Esox and Belone it is scarcely separated from the sacculus, in others as Pagellus, Scomber, Clupea, forming a considerable bursa-like swelling, and again in others, as Gadus, Lophius and Raniceps, flask-like and opening into the sacculus by a narrow tube.

In the Lophobranchii (Siphonostoma, Hippocampus) the sacculus is not separated into an upper and lower part, and this contrasts strongly with the disposition in Anarrhicas in which the two parts are nakedly distinct.

In connection with the canalis communicans or utriculo-saccularis Retzius finds, in confirmation of his previous researches and in opposition to the statement of Hasse, that in 11 of the 33 Teleostei described the communication exists. In Clupea and Corregonus it is just perceptible and in the remaining 20 it is entirely absent. In two thirds of these fishes there was present the macula acustica neglecta described by the author in a previous work, while it is absent in eleven. It is singular that in allied tribes such differences should exist.

The form and structure of the otolites would form an interesting subject if treated systematically; but the author has not been able to go into it farther than in connection with the other parts.

In the Elasmobranchs Retzius points out three very obvious types in the structure of the organ of hearing, viz. that of the Holocephali, of the skates, and of the sharks. The type of the Holocephali (Chimæra) forms an interesting transition to that of the Plagiostomes and through these to that of other fishes.

In all the Elasmobranchs the ductus endolymphaticus forms a pervious tube opening upon the surface of the head. In some it is straight and simple, in others it is narrowed or bent horizontally near its termination. There is also in all a separation of the recessus utriculi from the utriculus and from the outer and anterior ampullae, which is remarkable as compared with the form in Ganoids and Teleostei.

In Chimæra there is only a small opening, and the anterior and outer ampullæ open into the utriculus directly and not into the recessus. In the sharks there is often a pretty long tube between the utricle and the saccule, and there are in other instances interesting gradations in the development of this canal.

The macula acustica neglecta is present in all the examples of this group of fishes examined by Retzius. In Chimæra it constitutes a small nerve termination situated at the hinder end of the canalis utriculo-saccularis; but in skates and sharks the position is different, in consequence of the separation of the utriculus and the superior sinus. In the Elasmobranchs, in contrast with what is observed in the Teleostei and the sturgeon, the macula acustica neglecta is single or undivided.

As regards the lagena, this part is not separated from the sacculus in Chimæra, and its papilla is still in connection with the macula acustica of the sacculus. In the skates and sharks on the other hand the lagena forms a distinct bursa-like swelling of the sacculus, its papilla being in the skates only notched off from the macula acustica, but distinctly separated in the sharks.

In the sharks and skates, as is well known, the cartilaginous ear capsule is separated from the cranial cavity by a wall, but in Chimæra there is no partition so that it is a part of the same cavity with the brain.

In the Dipnoi there are interesting relations which receive their explanation from the study of the Elasmobranchs and particularly of Chimæra. In Ceratodus and Protopterus, and more especially the last, we find a very large recessus utriculi, not communicating directly with the utriculus or with the anterior or outer ampullæ, but united by an oval canal, (can. recessu-saccularis) with the sacculus, and through its intervention by the canalis utriculo-saccularis with the utriculus itself. The anterior and outer ampullæ open as in Elasmobranchs into the anterior end of the utriculus.

The single or undivided macula acustica neglecta lies in the base of the utriculus.

There is no distinct lagena cochleæ, but there is a separate papilla acustica lagenæ on the hinder and inner wall of the sacculus.

The endolymphatic duct was not found, and if it exists it certainly does not reach the surface of the head as in Elasmobranchs.

The utricle and its superior sinus are not divided vertically as in Plagiostomes. The ear cavity is, as in Teleostei, Ganoids and Chimæra, open to the brain cavity. The otolites consist only of small separate crystals. The ear of Dipnoi differs therefore considerably from that of Ganoids, and approaches more nearly to that of Elasmobranchs, especially of Holocephali or Chimæra. It seems as if the Dipnoi together with the Holocephali constituted a common but later divided lateral branch of the "principal philogenetic line." As regards the structure of their organ of hearing they cannot be placed, as was formerly attempted, in the same group with the Ganoids.

Between fishes and Amphibia there is of course in respect of the organ of hearing a gap not easily filled up; but yet not so great as might previously have been supposed.

In the lowest Urodeles the structure of the organ, as in Proteus, Menobranchus and Amphiuma, recalls forcibly that of Acipenser, although a separated lagena cochleæ exists in the Urodeles and there are other minor differences. They might be considered to have the properties of the Proganoids, which are inadequately represented, and the many intermediate members are unfortunately not known. With the divergent Dipnoi and Elasmobranchs on the other hand they have much less in common.

All the Amphibia, Urodeles as well as Anura, have the ear capsule closed on the inner or cerebral side by a partition wall. The lower Urodeles are especially characterised by the existence of a true perilymphatic space, limited by an external periosteal covering, and connected with the cerebral cavity by a tube (ductus perilymphaticus, Hasse) which passes through a special foramen. This space and its tubular outlet have nearly the same form in the higher Urodeles (Siredon, Salamandra), in which they were discovered by Hasse. The same observer also discovered the space and tubular outlet in the Anura, but in these animals the latter is dilated into a sack, and there also runs a long and somewhat complicated tube into the perilymphatic space where it opens.

In Urodeles and Anura, as in fishes, the ductus endolymphaticus proceeding from the sacculus goes, as shown by Hasse, through the apertura aquæductus vestibuli into the cerebral cavity, there to dilate into a comparatively large folded sack surrounding the brain but closed against the cerebral cavity.

The other morphological components of the membranous part of the organ of hearing in the lower Urodeles are the same as in fishes. There is a division into a superior and an inferior part, and these communicate with one another by means of a hole or tubular aperture, canalis utriculo-sacculus. Upon the projecting median side of this opening there is in the lower Urodeles a small nerve-twig and termination (derived from the ramulus ampullæ posterioris) lying as in Chimæra and to some extent in Plagiostomes on the wall of the posterior canal, and this Retzius regards as certainly corresponding to the macula acustica neglecta of Elasmobranchs and other fishes.

As regards the true cochlear part there is not, with the exception of the lagena and its papilla acustica which are present in all amphibia (except Cæcilia), any further place of nerve termination in the lower Urodeles, as Proteus, Menobranchus and Amphiuma; but in the higher, as Siredon, Menopoma, Pleurodeles, Salamandra and Triton, there is the first appearance of a separate papilla acustica basilaris cochleæ. But Retzius does not agree with Hasse and Kuhn in describing this as a true basilar part of the cochlea with its cartilaginous framework. But in the Anura he states that there is a well developed and distinctly separated pars basilaris cochleæ, which has been well known, since it was described by Deiters and Hasse, with the papilla acustica and cartilaginous rim or border, the thinner wall of which Retzius regards with Hasse as corresponding to the membrana basilaris. He has not succeeded in finding transition forms between Urodeles and Anura, and he does not believe that such exist.

Retzius describes shortly the accessory parts of the organ of hearing in Amphibia, into the details of which it is not necessary to enter here. He calls attention to the peculiar saccularly dilated duct (ductus fenestræ ovalis), which in the frog runs from the perilymphatic space to the cartilaginous plate of the columella and projects in the fenestra ovalis, and to the fact that a similar duct or diverticulum of the perilymphatic space is already more or less developed in the other Anura and the higher Urodeles.

Throughout the whole work Retzius has given minute descriptive details of the histological structure of the terminal nerve apparatus and other parts, all of which are illustrated by clear and appropriate figures drawn by himself from microscopic specimens. Space does not admit of our following the author in this description, and as he means to return to the whole subject of the finer structure of the membranous labyrinth when he has completed his second volume, we shall probably have another opportunity of reverting to it.

Retzius concludes his description with a reference to a "philogenetic line" or diagram which he has given, and in which the affinities of the several groups and genera are traced from the lowest point upwards and in diverging series. The principal line passes through the Proamphibia, Urodeles, Posturodeles, Proreptilia, and thence on to higher animals, and the successive lateral offsets from this line include the Myxinoids and Petromyzons, the Proganoids, Sturgeons, and Osteoganoids and the Osseous fishes on the one side; the Dipnoi, Proselachii, Holocephali, skates and sharks on the other, followed by Proteus, Siredon, Triton, and Anura.

ALLEN THOMSON.

LES PTOMAÏNES.—*Recherches chimiques, physiologiques et médico-légales.* Par I. GUARESCHI et A. MOSSO, de l'Université de Turin. Première partie. 8vo. pp. 55. Rome, Turin, Florence: H. Loescher.

THE organic alkaloids formed by the decomposition of the nitrogenous constituents of animal bodies, and to which the name of ptomaïnes has been given, have become of great importance from a medico-legal point of view. Our increasing knowledge of vegetable poisons has increased the possibility of new alkaloids which cannot readily be detected, being employed for the purpose of criminal poisoning. As alkaloids are formed, however, by the natural decomposition of the body itself, it would be quite possible that an innocent person might be convicted of poisoning from an alkaloid being found in a dead body, although no such had been given; and on the other hand a guilty person might escape on the plea that the poison actually found was a ptomaïne formed in the body and not administered at all.

In order to ascertain precisely the reactions and physiological actions of ptomaïnes, a committee has been appointed by the Italian Government to investigate the subject. The present work is the first-fruit of its labours, and the excellent way in which the subject has been worked out shows how wisely the committee has been selected. It would be here out of place to enter into the chemical part of the investigation, and we shall mention only those points that are of physiological interest. One of the first of these is the existence of organic alkaloids in different kinds of alcohol. E. Ludwig found trimethylamine in Austrian wine, and Krämer and Pinner found in alcohol a mixture of bases belonging to the picoline series, amongst others collidine. Guareschi and Mosso have also found in ordinary alcohol an organic alkaloid, probably either pyridine or a homologue of it. In amylic alcohol they found pyridine. It seems not impossible then that some of the injurious effects of amylic alcohol may be due to the action of organic bases contained in it.

From putrefied brain they obtained an extract which has an action on the motor nerves analogous to that of curara. It is, however, much less energetic. A much larger quantity of the substance is necessary to produce the same effect, and poisoning even when very complete lasts for a much shorter time than that of curara. Its action upon the heart is to render the pulsations stronger and slower. There is no period of acceleration, and the increase of energy lasts for a considerable time.

From putrefied fibrine they extracted a ptomaïne which produced in frogs complete immobility, muscular relaxation and loss of reflex, even when the muscles and motor nerves still responded regularly to stimulation by a faradic current.

A curious point was the agreeable odour which the poisoned frogs exhaled, resembling that of orange flowers. A finch poisoned by the ptomaïne vomited, trembled and showed diminished sensibility, but soon recovered. A rat weighing 76 grammes exhibited no symptoms after the injection of 20 milligrammes of the extract of putrefied fibrine. The difference between the activity of this ptomaïne and the ordinary organic alkaloids is very great, so that one may consider it as quite innocent in doses where coniine, nicotine or curara would be promptly lethal. Hydrochlorate of ptomaïne obtained from fibrine paralyzes the motor nerves like curara, but probably acts also on the muscles. When the muscle of a frog poisoned by it is irritated at regular intervals, it does not respond to each irritation by a contraction as an unpoisoned muscle would do. The contractions are sometimes high, sometimes low, and sometimes are absent. The excitability of the muscle becomes rapidly exhausted. This condition corresponds exactly to that described by Harnack in poisoning by lead and some other metals.

The similarity of the action of ptomaïne to curara leads the authors to discuss more particularly the action of curara itself, and they consider that the effect of this poison is not something *sui generis* but resembles the natural death of the organism. They found that the sciatic of an unpoisoned frog remains for a certain length of time at its normal level, and when it is irritated by opening shocks so feeble as to be hardly felt upon the point of the tongue a regular series of contractions is obtained from the muscle. This is the first stage. During this it is always easy to obtain regular contractions if the stimulus be sufficiently strong, but if the stimuli are very feeble, the contractions may neither be equal nor decrease regularly; they may be sometimes higher, sometimes lower, and even be wanting entirely.

In the second period, that of diminished excitability, this condition is still more marked, and in order to get regular contractions the stimulus must be increased in power. This second period may be observed in a frog which has been decapitated several hours previously or in one where the nervous excitability has been diminished by the influence of curara, of ptomaïnes, and of other nerve poisons. Whatever may be the poison employed, the result of poisoning by any drug which lessens the irritability of the nerves is exactly the same as that due to the natural death of the nerve. Under certain conditions Luciani has observed a curious irregularity in the beats of the heart which occur in groups, separated by intervals of quiescence. This takes place under the same conditions and is probably analogous to the irregularity in the contractions of voluntary muscles during the second state of dying, viz. that of decreasing excitability.

In the third period the nerve is no longer excitable by strong opening shocks which are isolated and separated by an interval of about a second, while it is still excitable by shocks which are more feeble but frequently repeated, like those of a Du Bois Reymond's coil.

This period is followed by the last, that of the death of the nerve, which completely loses its excitability, while the muscle still contracts on direct stimulation. Such is the course of the death of the sciatic nerve in the frog.

The action of poisons has nothing specific: they do not change the order of these four periods, they can only make them appear more quickly and accelerate the death of the nerve. The action of curara resembles natural death even in one point which has been supposed to be specific, viz., the continued sensibility of the sensory nerves after the motor nerves have lost their power. This condition is, however, produced, as the authors show, by simple compression of nerve trunks. When the lumbar nerves going to one leg of a frog are compressed for a short time by a small bull-dog forceps, irritation of that leg will produce movement in the rest of the body, while the leg itself remains paralysed and motionless.

Weir Mitchell has noticed that in wounded nerves the motor power may be abolished, while the sensory function is little altered. Erb has often seen in contusions of nerves sensibility re-established, while voluntary motor power was lost for ever. Lussana says that one characteristic symptom of rheumatic neuralgia is paralysis of the muscles and exaggerated sensibility of the corresponding parts.

The authors believe that it is this great vulnerability of motor nerves, and not an anatomical reason which furnishes an explanation of the readiness with which paralysis of the facial and laryngeal muscles occur in man, while disturbances of sensibility are comparatively very rare. The difference between the vitality of motor and sensory nerves becomes clearly evident from the effect of compression of the radial nerve which is one of the most common causes of paralysis of the muscles of the arm and of the hand; the paralysis being often complete while sensibility is hardly altered, if at all.

A study of other poisons supports the author's theory of the action of curara. A number of substances have been shown to have an action like it. Rabuteau showed iodide of tetramethyl ammonium; and Bufalini tetraethyl ammonium to have a similar action. V. Meyer obtained a similar substance from beer, and Bocci obtained another from human urine. In lead poisoning also the voluntary movements of the extensor muscles are abolished while sensibility is preserved.

The action of anæsthetics, which at first seems to be in opposition to this hypothesis, really agrees with it: thus in poisoning by chloral the posterior extremities preserve their sensibility after they are paralyzed, and even after the forelegs or head are paralyzed, changes in respiration show that sensory impulses are still nervously transmitted. The same thing has been noticed by narcotising with chloroform or ether, or by injecting pyridine or alcohol into the abdominal cavity. Even when the respiration has become too feeble to show any reflex change, the sensory nerves may be still showed to preserve their properties, for the pupil dilates at every irritation of the extremities.

The authors are no doubt right to a great extent in their views with regard to curara; and they may be also right to a great extent in considering that motor paths are sooner paralysed than sensory.

But if this be so, we can hardly do otherwise than assume that sensory cells are sometimes rendered functionally inactive before motor, as otherwise it is difficult to understand the relief of pain by morphia or anæsthetics while motor power still remains. It seems probable, however, that some drugs affect sensory sooner than motor fibres, for Pratt having surrounded the sciatic nerve of a frog with a paste of theine and water found that irritation of the left foot caused no reflex action while irritation of the spinal cord caused motion in both feet alike.

The curara-like action of ptomaïnes is a subject of great practical interest. In a paper on indigestion as a cause of nervous depression (Practitioner, vol. 25, pp. 265) the reviewer pointed out the resemblance between the symptoms of poisoning by paralyzing agents, and the muscular weakness and languor which occur in certain forms of indigestion, and drew attention to the fact that these symptoms of poisoning in indigestion might be due to substances absorbed from the intestine. At that time he was inclined to attribute these symptoms to peptones which have been shown to have a poisonous action, but the researches of Guareschi and Mosso appear to show that it may not improbably be due to ptomaïnes which have a curara like action. This seems all the more likely because not only has Bocci, as already mentioned, found a substance in the human urine which paralyzes frogs, but Bouchard, Rev. de Med. 1882, No. 12, has found ptomaïnes both in the urine of patients suffering from infectious diseases and in normal urine. He has also found them in large quantities in the fresh fæces of healthy persons as well as in diarrhœa and typhoid stools. The proportion of ptomaïnes in the urine to that in the fæces is as 1 to 40 or 50. These ptomaïnes he thinks are probably formed in the intestinal canal and absorbed into the blood. These researches afford an explanation of the acknowledged utility of purgation in nervous depression and especially of the utility of calomel which will not only remove ptomaïnes from the intestinal canal by its purgative action but will tend to prevent their formation as Wassilieff (Zeitschr. f. physiol. Chem. VI.) has shown it to possess a very considerable power of arresting fermentation and destroying micro-organisms.

T. LAUDER BRUNTON.

De la Démence Mélancolique (*On Dementia Melancholica. A Contribution to the Study of Periencephalitis, Chronic and Localized, and to the Study of Psychic Cerebral Localization*).—By DR. A MAIRET, Professeur Agrégé of the Montpellier Medical School. 296 pages and xi. planches lithographiées. Masson, 1883, Paris.

THE general idea contained in this book is the following:—Is it not possible to ascribe certain psychical symptoms which one may conveniently call *Dementia melancholica*, to functional disturbances and to diseases of the brain-cortex? M. Mairet, who, it must be said, is *médecin-adjoint* to an insane asylum, says he has often remarked among many insane some psychical disturbances, together with some limited anatomical diseases. These disturbances he ascribes to the diseases.

The disturbances are the following: melancholia of a delirous nature, and utter weakness of intellect which are both independent of one another. This melancholia may come on suddenly or slowly; it may be, for some time, the only symptom; but it may also be preceded or followed by the intellectual weakness. At the same time there are symptoms of cerebral disease.

The melancholia is generally of a lypemaniac character. In some cases lypemania is accompanied by much anxiety; that is, the patients think only of suicide, ruin, and so forth; in others the lypemania is *stupid*: the patients are foolish and stupid.

The symptoms of cerebral disease that are not of a psychical character are most generally the following. The face has generally a stupid look, brought on by relaxation of the facial muscles, together with redness brought on by a blood-stasis, on which M. Mairet seems to lay much stress. There is also some deviation of the muscles of one side of the face, some defect of symmetry in the mouth, and a little hesitation in the speech. M. Mairet insists very much upon another symptom; that is, on ptosis of the eyelids, either on one or both sides. Other symptoms are to be found in the limbs: formication, feeling of heaviness, for instance; others concern the head: such are vertigo, apoplectiform attacks, cephalagia, heat of the head, and other such. As to the cerebral lesion which M. Mairet considers as the cause of the symptoms alluded to, it is a *localized periencephalitis* of which the seat is the *inferior aspect of the brain*, either on the *whole surface* or on a *limited region of that surface.* M. Mairet considers this disease as brought on, either by propagation of an inflammatory disease of the eye, or ear, or other organ or tissue in the neighbourhood, or by general depressing causes, physical or mental.

Such is the author's general theory, which he supports by many cases with full particulars. But M. Mairet has wished to know whether experiments on animals could in some way support the idea. He has accordingly studied on dogs the effect of certain experimental lesions, observing whether there was some peculiar sadness to be detected on their countenance after the operation. It must be confessed that this part of the book of M. Mairet is much less reliable and valuable than the preceding one. Even if the operation be conducted in the very best manner, it seems very difficult to tell with some degree of certainty whether the animal is "triste" or not. M. Mairet pours upsome water with hydrochloric acid on the inferior aspect of the brain, and watches whether the dog becomes triste. If we judge of the mental condition of the dogs by the sketches M. Mairet gives in his book, they certainly seem very triste; but might they not seem as much so if one had only made a hole in their head without sending any hydrochloric acid into it; or might they not seem exactly the same if the hole had been made on the superior aspect of the head, and hydrochloric acid poured in, *secundum artem?*

M. Mairet has conducted three experiments on dogs, and concludes that the sphenoidal lobe of the cerebrum seems to be the centre for sadness. We cannot agree with the author's views; three experiments are not enough, and M. Mairet ought to study the influence of other brain-lesions on the *genesis* of sadness, in man as well as in dogs. His book is nevertheless a very interesting and able effort to solve a very difficult problem of brain-function. It is well written, and students of neurology should read it.

HENRY DE VARIGNY.

De la Chute et de la Dystrophie des Ongles chez les Ataxiques. Par E. DOMECQ-TURON. Thèse pour le Doctorat en médecine. Bellier & Cie. Bordeaux, 1883.

THIS is an interesting contribution from the clinical side towards a further knowledge of the trophic lesions of tabes dorsalis. The author has collected fourteen cases—some of them already published by MM. Joffroy, Pitres, Pouget, and Roques, others communicated to him by his friends—which render it highly probable that occasionally loss and deformity of the toe nails are real trophic lesions which should be placed in the same category as the joint affections, and the perforating ulcers of the feet which are not uncommon in the course of this disease. His cases seem to establish the following facts: that loss of the nails is usually limited to the great toes, that it has no fixed relation to any particular period of the disease, and may occur over and over again. The conditions which precede the nail shedding are very variable: without any history of injury the matrix of the nail may sometimes become ecchymosed, and the neighbourhood of the nail may be anæsthetic, or be afflicted with abnormal sensations, or even intense pain. The nail may be reproduced either of normal shape, or more or less deformed. The description, in fact, which the author gives of the nail appearances before shedding reminds one forcibly of the trophic changes which are so often seen when there has been injury to a peripheral nerve, and neuritis is kept up by irritation at the cicatrix. It is this similarity which more especially leads to the supposition that loss of the nails is probably due to a peripheral neuritis rather than to any lesion in the spinal cord, but in any case, it seems clear that spontaneous shedding of toe nails is more often observed in tabes dorsalis than in any other of the degenerative diseases of the marrow.

Not less interesting than loss of the nails are the nail deformities —the *Dystrophie*, as he calls it—of which the author treats in the second half of his thesis. Here again we are reminded how often the nails may become crumpled or misshapen after lesions of peripheral nerves, and it is well known how the nutrition of the nails is dependent on bodily health, and that constitutional illnesses may bear traces of their severity in transverse furrows of all the nails

The dystrophy of the nails in tabes dorsalis, however, is usually limited to the nails of the toes—more especially of the big and little toes—limited, in fact, to the limbs which are most frequently affected with lightning pains or other abnormal sensations. In all the cases recorded the finger-nails presented nothing abnormal. This important association is, moreover, established that at whatever period of the disease the deformity arises, whether it affects the great toe only or the other toes as well, it coincides with the occurrence of severe pains, or with sensations of numbness and creepings in the toes. With regard to the deformity itself, the nails become thickened, elevated or crumpled, they develop longitudinal or transverse furrows, are raised from their natural beds, become intensely hard, and whatever be their colour, lose their natural transparency. In some instances these changes have come on with great rapidity, and once established they seem to last an indefinite time; and as of the spontaneous shedding, so of these dystrophic alterations, the conclusion seems to be that the underlying cause is a peripheral neuritis. The author's thesis is of especial value, because of its well-recorded cases, and the appearances described by him are shown in two plates of coloured drawings. Many observers must be familiar with the kinds of nails depicted, but important results will flow from M. Domecq-Turon's contribution to the clinical history of tabes dorsalis, if the existence of these nail deformities leads to the more careful examination of all patients who present them, and tabes dorsalis be thereby found when least expected, or when, as so often happens, the more prominent symptom of ataxia is entirely absent.

HERBERT W. PAGE.

Critical Digests and Abstracts from Journals.

Report on Visceral Neurology. The Vagus.—The whole subject of the pathological relations of the vagus has been taken up by Kredel, assistant to Professor Riegel in the Medical Clinic of Giessen, and elaborately treated in his inaugural dissertation (*Deut. Arch. f. klin. Med.* xxx., p. 547). The first part of the work touches very lightly on the various diseases and disorders connected with the branches of the vagus to the heart, the larynx, and the lungs proper. The nervous theory of asthma is well summarised; but to this part of the subject we shall immediately return when we consider the researches of Kredel's master, Professor Riegel. Angina pectoris is next dealt with, much after the arrangement of Eulenberg and Guttmann. All these summaries are but an introduction to the special subject of the work, which is devoted to the discussion of two cases under the writer's observation, apparently of an anginal and asthmatic nature combined. As is well known, the respiratory disturbance in true angina pectoris is insignificant, and has been referred, such as it is, to the pain. Kredel contends, however, that a careful perusal of recorded cases reveals the incorrectness of this view; and his own cases, in his opinion, support the belief that the respiratory and cardiac branches of the vagus may be at one and the same time the seat of a severe neurosis. The best-marked case may thus be described. A girl of 16, suffering from mitral incompetence, on being sent to bed for the purpose of examination, was suddenly seized with palpitation and urgent dyspnœa, the pulse rising to 148 and the respirations to 52. Under the writer's observation the dimensions of the pulmonary resonance rapidly increased, along with the size of the heart as tested by the impulse. In the course of half an hour, the urgent symptoms continuing, cough set in with expectoration of blood-stained phlegm, and therewith râles were discovered for the first time in the chest. The dyspnœa, pulmonary dilatation, and cardiac frequency had not entirely disappeared at the end of 18 hours. There was no relapse. Kredel's interpretation of this attack is that it was a combination of inhibition of the cardiac branch of the vagus, spasm of the bronchi from irritation of the bronchial branches of the vagus, and consequent congestion of the mucous membrane of the respiratory passages. He confesses that the frequency instead of depth of the respiratory acts was not suggestive of true spasmodic asthma, but the acceleration of the pulse and the bronchial catarrh might account for this peculiarity. The whole condition, in a word, was, he believes, a neurosis of the vagus originating in its trunk. He has difficulty in settling—indeed, he confesses he cannot determine—how a lesion of the trunk of the vagus is likely to cause at once paralysis of the cardiac branches and irritation of the pulmonary branches. Unfortunately for Kredel's elaborate theory; we fear that in this country the condition which he observed and has described with such praiseworthy accuracy would be considered to be nothing more than temporary pulmonary congestion of purely mechanical origin.

The neurosal theory of *bronchial asthma*—that is, the relation of disorder of the vagus to this affection—is the subject of a very elaborate investigation, involving a large number of experiments on animals, by Professor Riegel of Giessen, the author of the article on Asthma in Ziemssen's *Handbuch*. The paper (which appears in the *Zeitsch. f. klin. Med.* v., p. 413) commences with an exceedingly valuable *résumé* of the arguments for and against the spasmodic theory proper of asthma. Riegel insists, in the first place, upon our keeping perfectly distinct the two questions as to whether, first, the bronchi contract when the vagus is stimulated, and whether, secondly, bronchial asthma is due to irritation of the vagus. The first question may now be considered as definitely settled in the affirmative—that faradisation of the vagus does increase the pressure within the bronchi, and that this rise of pressure is due to contraction of the bronchial muscles. Riegel's experiments, beginning with this part of the question, were arranged to discover, next, whether the lung increased in size under these circumstances. The result was positive, the dilatation being rapid and considerable, lasting 10 minutes, and slowly disappearing. These observations were made on the undivided vagus, and now came the difficulty—by what route was the impression conveyed which ended in pulmonary dilatation? It was first determined that the irritation did not act peripherally; and the important point appeared to be definitely settled, that, after all, the dilatation of the lungs in asthma is not due to spasmodic contraction of the bronchi. A further series of experiments revealed the fact that irritation of the vagus produces asthma reflexly, that is, by a centripetal impression which passes through the respiratory centre and reaches the diaphragm, and that the dilatation of the lung is almost entirely due to sudden inspiratory depression of this muscle, followed by its tonic contraction. The theory of bronchial spasm as the cause of asthma must, therefore, be given up as being insufficient. There is unquestionably a rise of pressure in the respiratory passages when the vagus is irritated, as we have seen, but it is much too small to account for the dyspnœa of asthma, and is quite insignificant in comparison with the reflex effect through the diaphragm. Riegel, however, wishes it to be distinctly understood that he does not publish these results as a full and complete explanation of the phenomena of bronchial asthma, inasmuch as vaso-motor disturbances and bronchial hyperæmia are probably elements in the attack.

Respiration.—Some account has been given from time to time in these *Reports* of the discussion (of a thoroughly practical kind) which has been going on in Germany, respecting the physiology of the respiratory branches of the vagus, including the effect of artificial stimulation of the trunk of the nerve in the neck, and of mechanical irritation of its pulmonary roots by respiratory movements. Knoll, who is well known for his important contributions to the physiology of the circulation, has recently published several observations on the effect of electrical and mechanical stimulation of the cervical vagus on the respiration. In the first place, he has shown that modifications of the proper nerve current (*Eigenstrom*) in connection with the transverse section of the central stump of the trunk, can be induced by manipulation, and that these modifications exert a distinct effect upon the respiratory acts in expiratory time. The peripheral stump gives no corresponding reaction. Secondly, Knoll's experiments appear to prove that the primary effect of faradisation of the central stump is expiratory, the more familiar inspiratory effect following stronger stimulation only. But even whilst inducing continuous contraction of the diaphragm in this way, faradisation of the cervical vagus causes a certain amount of simultaneous inhibition of the respiratory movements; for the muscles of extraordinary respiration are relaxed, there is no respiratory movement in response to the cerebral anæmia, and reflex respiratory movements do not follow stimulation of other afferent nerves (*Wien. akad. Sitz.* LXXXV. and LXXXVI.; and *Cent. f. d. med. W.*, 1883, No. 22).

The question of the relative influence upon the respiratory centre of an excess of carbonic acid and a deficiency of oxygen in the blood, has been revived by Bernstein (Du B. Reymond's *Archiv*, 1882, p. 313: and *Cent. f. d. med. W.* 1883, p. 354). This observer concludes that "carbonic acid dyspnœa" is chiefly expiratory in character: the dyspnœa caused by want of oxygen chiefly inspiratory. It is not until the controlling function of the vagus has been removed, by section of the nerve in the neck, that these points can be demonstrated.

Nervous Influence of Respiration upon the Circulation.—One of the most complex problems in the physiology of the circulation is the effect of respiration upon the movement of the blood in the lungs, the heart, and the systemic vessels. Whilst the phenomena themselves are but imperfectly known, the manner in which they are produced is necessarily still more obscure. Manifestly a great part of the influence of the movements of the chest and lungs upon the circulation is purely mechanical, but there is as unquestionably an abundant nervous connection also between the two great functions. A contribution to this part of the subject has just been made by Taljanzeff (*Cent. f. d. med. W.*, 1883, p. 401), who has discovered that faradisation of the central end of the pulmonary branches of the vagus, divided where they are passing to the (left) lung, near the bronchus, causes great slowing of the heart, increased size of the pulse, and marked fall of the aortic pressure. As these effects can be arrested by section of the corresponding vagus in the neck, the impression manifestly travels to the medulla through this route. It would thus appear that the movements of respiration modify the general blood-pressure through the centripetal branches of the vagus in the lungs.

THE HEART.—Perhaps no portion of morbid anatomy is so much a blank as the diseases of the nervous structures of the heart. Dr. Uskow has investigated with care the condition of the nerve-fibres and ganglia in a certain number of instances of enlargement and other morbid states of the heart, and has arrived at the following conclusions (Virchow's *Archiv*, Band 91, III., p. 453). In hypertrophy of the heart the cardiac nerves lose their medullary sheath, and undergo a process of nuclear proliferation. In unilateral hypertrophy of the heart these changes may be found to be confined to the corresponding side. In certain acute diseases the cardiac nerves undergo parenchymatous inflammation. The only changes which Uskow could discover in the nerve-cells was thickening of the capsules and nuclear proliferation, the protoplasm never presenting any appreciable alteration.

VASO-MOTOR NERVES.—The vaso-dilator function of the sympathetic is the subject of an elaborate investigation by Dastre and Morat (*Arch. de Physiol. Norm. et Path.* 1882. ix. 2 and 3; and *Cent. f. d. med. Wiss.* 1882, p. 731.) The following are the most important conclusions, as far as our present purpose is concerned: 1. Weak induction currents applied to the upper end of the divided vago-sympathetic trunk in the dog, causes active congestion of the mucous membranes and skin of the same side, with elevated temperature and swelling. The dilatation of the vessels is a direct effect, and not the result of exhaustion of the nerves. It is the sympathetic alone that contains the vaso-dilator fibres, just as it also contains vaso-constrictor, secretory and ciliary (dilator) fibres. 2. The vaso-dilator fibres of the trigeminus (superior maxillary) probably come from the sympathetic. 3. The centre or centres for this function (as regards the mouth) are situated in the dorsal part of the cord (cornua); and the route from the centres to the cervical sympathetic passes through the *rami communicantes* from the 1st to the 5th dorsal. 4. Cerebral conditions, such as emotions, readily influence these nerves. 5. The vaso-dilator nerves of the head are also very amenable to reflex influences, of which those that traverse the vagus are the most powerful: such influences, for example, arise in the outlying regions of the vagus (above all, in the respiratory organs), travel through the medulla and upper cord, pass into the cervical sympathetic, and thus reach the face. 6. Occasionally vaso-constriction and not vaso-dilatation is the result of weak stimulation of the cervical sympathetic; but a different set of nerves are then in action.

TROPHIC NERVES.—A very elaborate contribution to this subject, of a purely experimental kind, by Lewaschew, of St. Petersburg, working under Professor Botkin, appears to confirm the growing belief that the nutritive changes which follow nervous lesions are referable to vascular disturbance (*Cent. f. d. med. W.*, 1883. p. 193). In one most important respect Lewaschew's conclusion is different from previous views: he holds that the dilatation of the vessels and associated phenomena, consequent on nervous lesions, and frequently the precursors of "trophic" changes in the tissues, are due to irritation of the vaso-dilator nerves, and not to paralysis of the vaso-motor nerves. Lewaschew's investigations consisted in irritating the sciatic nerve in dogs, by means of threads steeped in weak acids or salt, and then noting the phenomena that ensued in the limb, as well as making careful post-mortem examinations of all the parts involved. Contraction of the vessels of the limb was rarely the result of this operation on the sciatic; and when this result was obtained, no trophic changes ensued. Generally the very opposite condition of circulation was produced: the vessels dilated and pulsated, the limb swelled, the temperature rose; and all in the course of a few days. These phenomena would persist for several months, and then disappear rather quickly, but meanwhile more permanent changes were taking place. The soft parts round the nails were swollen and ulcerated, the epidermis of the sole thickened, the bones became enlarged, the electrical condition of the muscles was altered. Ultimately, the limb generally became atrophied, with diminished temperature; the skin and some of the muscles were reduced in volume; the subcutaneous tissues were sclerosed and white; and the blood-vessels presented contractions and dilatations of their lumen. Microscopical examination confirmed the observation of primary vascular dilatation, and secondary sclerosis around the vessels. In some of the experiments, excess of irritation caused muscular paralysis of the limb, complicating but practically confirming the observation. The absence of paralysis and anæsthesia in the best cases suggested the absence of vaso-motor paralysis, and the presence of vaso-dilator irritation. The variety of results obtained confirmed this view; as well as the rapidity of their development, and the degree to which they advanced.

Two very definite cases of "trophic" or "neuro-paralytic" keratitis may be here recorded. In the one, under the care of Professor Senator, ulceration of the left cornea was associated with trigeminal paralysis, although the "spontaneous" movements of the lids were perfect, as well as the reflex from the opposite cornea. Senator's view is in favour of the "vaso-motor-trophic" origin of such a case. The second case, under Kahler, commenced with paralysis of the right facial nerve only, without keratitis; the lesion being a tumour involving the right ganglion geniculi. As the disease spread, sensibility disappeared in the region of the right trigeminus; and thereupon the cornea very quickly ulcerated, although it had been most carefully protected during the whole illness.

Herpes.—Nieden has described a remarkable case of recurrent herpes in the region of the ophthalmic division of the left trigeminus, after lesion of the cervical spine. A man who had sustained an injury in the neighbourhood of the superior cervical ganglion, and who for the next six years had suffered from severe attacks of cephalalgia and ciliary neuralgia, became subject to eruptions of herpes in connection with the left eye. The cornea, and the skin corresponding to the supra-orbital and supra-trochlear nerves were the seats of the herpes, whilst the nasal area was spared. During the next six years the eruption recurred four times, and, as in the first attack, it was always accompanied by paresis of the vessels of the left half of the face, periodical headache, hyperæsthesia of the skin, and palpitation of the heart. No doubt the left cervical sympathetic was paralysed, with consequent neurosis of the first division of the trigeminus.

Trophic changes in the skin, hair, or nails were entirely absent in a case of injury to the shoulder and brachial plexus in which there was extensive paralysis of the muscles of the arm, forearm, hand, and chest (Erlenmeyer, *Cent. f. d. med. Wiss.* 1883, p. 112). On the other hand, fresh instances continue to be reported of affections of the toenails in locomotor ataxy. In one case recorded by Roques (*Union méd.* 1882, No. 91) the loss of both great toenails was preceded three months by continued subungual ecchymosis, and the corresponding region of the skin was anæsthetic.

THE STOMACH.—Prof. Leyden, of Berlin, has published a long and valuable paper upon periodical vomiting of nervous origin, more familiarly known as "gastric crises" (*Zeitsch. f. klin. Med.* iv., p. 605). Whilst most common in association with locomotor ataxy, periodical nervous vomiting may occur in other diseases of the cord, and occasionally appears to be independent of all spinal lesions, although complicated by other nervous symptoms. This statement is, of course, made with full knowledge of the important observation of Charcot that gastric crises may occur as the earliest symptom of ataxy, sometimes even years before the difficulty of locomotion. The onset of these periodical attacks is sudden; and the principal symptoms are severe copious vomiting, epigastric pain, a feeling of severe illness, intense headache, hyperæsthesia of the special senses, and sometimes disorder of the mind. The attack may last two or three days; then a little food is taken; the appetite and digestion are rapidly restored; and in a few days the patient is quite well. As important concomitants of the more striking symptoms, must be mentioned retraction of the abdomen, obstinate constipation, scantiness of the urine, frequency and fulness of pulse without pyrexia, and occasionally severe pains in the limbs. Leyden has twice had an opportunity of making a careful *post-mortem* examination in this class of cases, with entirely negative results as far as the stomach was concerned. The treatment consists in general and local rest, including the use of morphia.

Diabetes and Polyuria.—A case in which the tracts between the "diabetic centre" and the viscera were involved, rather than the centre itself, is recorded by de Jonge in the *Archiv f. Psych.* XIII., 3, p. 658, and *Cent. f. d. med. W.*, 1883, p. 335. A man of 37, with phthisis and tubercular peritonitis, died of sudden right hemiplegia. Post mortem, a bean-sized tumour was found in connexion with the medulla, beginning 0·2 mm. below the left olivary body, extending to the first cervical nerve, and involving the substantia gelatinosa, beyond which it slightly extended on all sides.

A less definite instance of the connection between lesion of the central nervous system and polyuria was observed by Hagenbach (*Jahrb. f. Kind.* n. f. XIX., p. 214; and *Cent. f. d. med. W.*, 1883, p. 352). A child of 4½ years, who had passed at times as much as 10 litres of urine, with steady emaciation and great thirst for nine months, died of tubercular meningitis. Besides the usual post-mortem appearances, a caseous mass was found involving the infundibulum, and a spot of softening in the corpus striatum, with dilatation of the lateral and fourth ventricles.

The Bladder.—The movements of the urinary bladder, and especially the influence of nervous impressions in urination, have been studied by Mosso and Pellacani (*Arch. ital. de biologie*, 1882, i. 1; and *Cent. f. di. med. Wiss.* 1882, p. 705). Besides the movements communicated to the bladder by the respiratory acts, there are purely reflex vesical contractions, which are readily distinguished from the communicated movements by their duration, the shortest of them lasting 6 or 7 seconds. These reflex movements are induced by sensory and psychical stimuli; for instance, a loud noise or a powerful emotion; and, indeed, the authors go so far as to say that "every mental act in man is accompanied by a contraction of the bladder." And not only this, but one can voluntarily empty the bladder without employing the respiratory muscles at all. It is probable that the "voluntary" power referred to in this connection is the power of the will to call up emotions or ideas, which in their turn induce visceral movements such as are familiar in connection with the rectum, sexual organs, and circulatory apparatus. Mosso and Pellacani indeed show that the reflex movements of the bladder correspond closely with those of the arteries, whilst they are quite independent of them. (Cf. 'Brain,' April 1881 and July 1882).

The Uterus.—Frommel has arrived at some novel conclusions with respect to the movements of the uterus (*Zeitsch. f. Geb. u. Gyn.* viii. p. 205; and *Cent. f. d. med. Wiss.* 1883, p. 418). He finds that these movements are not referable to a uterine centre lying without the organ. Its contractions are spontaneous, and may occur rhythmically in all stages of its development. Disturbances of the circulation have a marked influence upon the uterine movements, compression of the aorta completely arresting them, and compression of the inferior cava producing the same effect, though less quickly. Fall of the body temperature slows the contractions without interfering with their energy; elevation of the body temperature accelerates them, but high fever completely arrests them. The results apply to rabbits only.

REFLEX PARALYSIS.—Several instances are on record of paralysis of a limb in connection with pleurisy, mostly after removal of the exudation, or in consequence of washing out of the pleural cavity. Francotte has recently described a case in which paralysis of the right arm occurred during the spontaneous disappearance of a moderate empyema of the same side. The paralysis was but slight and transitory. In the opinion of Francotte it was reflex in its nature, the seat of irritation being the sensory nerves of the pleura (*Cent. f. d. med. Wiss.* 1883, p. 109).

J. MITCHELL BRUCE, M.D.

Intermittent Spinal Paralysis of Malarial Origin. BY V. P. GIBNEY, M.D. (*American Journal of Neurology and Psychiatry*, Feb. 1, 1882.)—Two cases are given, occurring in children of 6 and 7 years old, living in New York. In the first case, there were four separate attacks of paralysis, affecting the lower limbs each time, and once the upper limbs also. They were preceded by fever; malaria was suspected in the house where the child lived. Electrical reactions of degeneration, and wasting of some muscles were observed; so that the case simulated infantile paralysis. Recovery took place within times varying from a week to six months, being aided by removal from home and the administration of quinine.

In the second case there were three attacks, each preceded by fever, during which pains in the limbs had been a prominent symptom. Two weeks after the last attack the child had convulsions, and finally died of exhaustion. No post-mortem.

A third case is mentioned, of a child born in a highly malarious place, of a mother who herself was suffering from malaria. The child in its first year had symptoms of ague, in its second an attack of hemiplegia preceded by chills and fever; when examined by the author more than six weeks later, the hemiplegia had partly recovered, but both arms were tremulous.

In all three cases the father of the patient was intemperate and the mother delicate. The spleen was not enlarged. On one occasion desquamation of the skin was observed.

As causes of the paralysis, the author thinks that he can exclude —lead, from the distribution of the paralysis—hysteria, from the fact that the electrical reactions were abnormal—infantile paralysis, from the complete recovery made from most of the attacks.

Assuming malaria to be the cause, he suggests that the pathology of the disease may consist in a congestion and œdema of the cord, analogous to that of the spleen.

Case of Destructive Lesion of the Cuneus, accompanied by Colour-blindness. BY N. E. BRILL. (*American Journal of Neurology and Psychiatry*, August 1882.)—A man, ætat. 63, had a sudden attack of coma, lasting two days, followed by incomplete right hemiplegia and right hemianæsthesia. In a few weeks there appeared hyperæsthesia of the right thigh, tremor of the paralysed side, tinnitus, diplopia, and notably colour-blindness. He appears to have been unable to recognise blue or green. There was no limitation of the visual field. No ophthalmoscopic examination was made. There was a certain amount of aphasia, most marked with respect to the names of colours.

Rigidity with tremor set in on the right side, also some tremor of the left thumb. Patient sank in a few weeks more.

Post-mortem.—The large arteries at the base of the brain were found to be atheromatous. There was a well-defined patch of softening on the left side, involving the cortex of the left occipital lobe where it borders on the calcarine fissure, the upper edge of the ingual gyrus, and the lower half of the cuneus. The white substance was involved to a depth varying from 3½ to 1 centimetre.

Case of three Tumours of the Encephalon. BY W. R. BIRDSALL, M.D. (*Journal of Nervous and Mental Disease*, April 1882.)—Female, ætat. 20. Headache, followed by a chill and fever, six months before admission. Headache mostly occipital ever since. Paralytic symptoms of two months' duration, with the exception of atrophy of left half of tongue, which was said to have always existed. Vision blurred from childhood. Diplopia, and tinnitus (left ear) for three weeks.

On examination, complete facial paralysis, right side; paresis of right sixth nerve. In left hand some athetesis, but not much loss of power: in left leg, considerable lose of power, with increase of tendon-reflex. Double optic neuritis.

Later on, rapid failure of vision, vomiting, dysphagia, increased difficulty of articulation: convulsion of left arm just before death.

Post-mortem.—Tumours were found in the following situations:

(1) In the right hemisphere, encapsuled in the pia-mater overlying the upper half of the ascending frontal convolution, the upper two-thirds of the ascending parietal, the whole superior parietal lobule and part of the inferior. These parts formed a sort of cavity in which the tumour lay: they were thinned by pressure, but not destroyed.

(2) At the junction of the pons with the medulla, upon the lower surface of the cerebellum, and attached to the seventh nerves of the left side. These nerves ran over its lower surface, the facial was not involved, the auditory was attached to it, part of that nerve running through, part being spread over the tumour.

(3) In the medulla oblongata, reaching from the level of the lower third of the fourth ventricle to below the calamus scriptorius, situated nearly in the centre of the medulla, but involving the left side more than the right. The cells of the left hypoglossal nucleus were shrunken and diminished in number. Otherwise the nerve-tissue was for the most part normal, being pushed aside rather than destroyed.

The character of the tumours was that of fibro-sarcoma.

The author remarks that the left hemiparesis can be accounted for by the tumour of the right hemisphere; the tinnitus by the tumour of the left auditory nerve, the atrophy of the left half of the tongue, with the dysphagia and difficulty of articulation by the lesion of the medulla; but for the paralysis in the district of the right facial and right sixth nerves he can assign no cause.

The Paths of the Vasomotor, Sudorific, and Sensory Nerves in the Spinal Cord. By Isaac Ott, M.D. (*Journal of Nervous and Mental Disease*, July 1882.)—These experiments have reference to the nerves of the cat's hind-limbs, and were made on the plan of previous experiments (on the rabbit) by Ludwig and Nicolaides.

If a hemisection of the cord be made in the dorsal region, the feet become vascular: if now the cord be stimulated in the cervical region, both feet become pale; it is therefore inferred that vaso-motor fibres run in both halves of the cord.

After complete section, stimulation of the cervical region produces no effect; hence it is in the cord, and not in the abdominal sympathetic that the vaso-motor fibres run.

Further, since sweating of both feet follows hemisection of the it is inferred that the sudorific fibres follow the same course vaso-motor.

espect to the sensory fibres, rise of arterial pressure was an index of effective stimulation of them. If the whole cord was divided, irritation of the sciatic nerve produced no effect. If either lateral column was left intact, irritation of the sciatic caused a rise in blood-pressure, which took place even if the grey matter were destroyed. If both lateral columns were divided, no rise in blood-pressure could be obtained by irritating the sciatics. It is therefore inferred that the sensory fibres run in the lateral columns. If a hemisection was made, irritation of the corresponding sciatic had nearly the same effect as irritation of the opposite sciatic; therefore the sensory like the vaso-motor fibres decussate in the cord.

The author's conclusions are:

(1) That the sensory fibres decussate in part in the spinal cord.

(2) That the vaso-motor fibres also do.

(3) That the sudorific fibres follow the vaso-motors and decussate.

(4) That the vaso-motors run in the lateral columns.

On the so-called Family or Hereditary form of Locomotor Ataxia. By Wm. A. Hammond, M.D. (*Journal of Nervous and Mental Disease*, July 1882.)—The author recapitulates the six cases originally reported by Friedreich. Three new cases by Friedreich and one by Carre are alluded to. Twelve new cases are then given. None of all of these were in the author's opinion instances of true locomotor ataxy, meaning by that term primary sclerosis of the columns of Burdach. The symptoms presented in the hereditary disease are weakness (true paralysis?) beginning in the lower limbs, advancing upwards and finally causing embarrassment of speech. Though inco-ordination may be present, the gait is not that of true locomotor ataxy. There are no lancinating pains; indeed pain may be absent altogether. There is no sense of constriction round the waist. There is no loss of cutaneous sensibility and micturition is natural; the pupils are unaffected. [The patellar tendon reflex where mentioned was abolished.]

The author thinks that the primary lesion is to be found in the medulla, possibly also in the cerebellum. If the spinal cord be implicated (as it certainly was in Friedreich's cases), th posterior columns are not the only, nor even the principal seat the disease.

Case with Tumours in the Fourth Ventricle, u panied by special Nervous Symptoms. By H. D M.D. (*Journal of Nervous and Mental Disease*, July 18 patient, a negro, ætat. 30, who had had secondary syphili acute pneumonia and pericarditis, without exhibiting any sy of intra-cranial tumour.

Post-mortem, there were found:

(1) A lobulated tumour measuring 25 mm. × 13 × 10, attached by a ribbon-like pedicle to the posterior surface of the right restiform body, pressed upon from above by the right tonsil of the cerebellum, and filling up a considerable portion of the fourth ventricle.

(2) A smaller, measuring 8 mm. × 3, situated similarly on the left restiform body, but without a pedicle.

The tumours were very vascular; their microscopic structure was that of gliomata.

The medulla was tough, like india-rubber, but its minute structure was natural.

The author remarks on the symmetry of the tumours, and on the absence of symptoms, though he raises the question whether the pneumonia may not have had something to do with pressure on the pneumogastric nucleus.

Two Cases of Diphtheritic Spinal Ataxia. By Drs. Julius Rudisch and George W. Jacoby. (*Archives of Medicine*, December 1882.)—Two cases are given from the author's own observation illustrating a form of diphtheritic paralysis resembling locomotor ataxy.

Case 1.—Male, ætat. 20. Mild attack of diphtheria; two weeks afterwards, transient paralysis of soft palate; next, weakness of legs, then of hands. In lower limbs, there was some anæsthesia of the soles, ataxic gait, unsteadiness on closing eyes. In upper limbs, retardation of sensation, want of precision in fine movements. Patellar tendon reflex absent. Electro-contractility of muscles normal. No cerebral symptoms. In rather more than a year, all the symptoms had disappeared, except that the patellar-tendon reflex was still absent. In a year more this also had returned.

CASE 2.—Male. Diphtheria, followed in two weeks by paralysis of the palate. In three weeks more, severe shooting-pains in legs and arms; then unsteadiness of gait. When seen, his gait was ataxic, he could not stand with his eyes shut, or walk in the dark. Fingers numb, fine movements of hands difficult. Patellar tendon reflex absent. Action of pupils normal. Electro-contractility of muscles normal. In seven months he completely recovered, and the patellar tendon reflex had returned.

A third case is quoted from Rumpf.

From the symptoms, the authors conclude that the posterior columns were affected, though on reviewing the investigations that have been made into the pathological anatomy of diphtheritic paralysis, it appears that actual lesion of this part has not as yet been found post-mortem.

Case of Myxœdema. BY ELIZABETH M. CUSHIER, M.D, (*Archives of Medicine*, Dec. 1882.)—Female, ætat. 57. Her general health had fallen off since nervous shocks in 1861. Within a few years after that time, swelling of the face was noticed, inactivity, slight deafness. When first seen by author (1877), the legs were œdematous, the tissues of the face thickened, the eyelids baggy, suggesting Bright's disease, but there was no albuminuria.

She improved under tonics and diuretics.

After two years there was a return of the œdema in the legs, vesicles formed and became inflamed and ulcerated: the walk was slow and clumsy; she was slow in rising from the chair or lifting her head. No anæsthesia.

The face gradually became more characteristic; the aspect expressionless, the cheeks firm, waxy, rosy pink in colour: the skin round mouth and eyes white and transparent; the lips motionless. Temperature subnormal, pulse slow.

Subsequently the cardiac dulness became much increased in area: the abdomen swelled. She died in June 1882, after an attack of facial dermatitis, and inflammation of the left upper arm.

Post-mortem.—The subcutaneous tissue of the neck seemed infiltrated with transparent material: the heart was hypertrophied, the kidneys rather large. In all these organs there was seen, microscopically, a swollen state of the connective tissue, and an infiltration with small, spherical, highly refractive bodies. The normal histological elements were atrophied. In the thyroid, which was small and undergoing (in the left lobe) cystic degeneration, it was found that the alveoli were filled with hyaline material and the blood-vessels were infiltrated.

The changes in the spinal cord were as follows:

Vessels.—Their walls were thickened and infiltrated with hyaline substance; the vessels themselves were dilated, and there were numerous small hæmorrhages, many of them old.

Nerve-cells.—These had undergone yellow degeneration, or were atrophied, or had lost their processes. These changes were most marked in the lumbar region: they affected both the anterior and posterior cornua.

White substance.—Showed small spaces filled with hyaline material; here and there the nerve-fibres were atrophied.

The author remarks on the limitation of the mucoid œdema to the skin of the face and neck; and upon the paresis of the lower extremities. This latter she explains by the lesions in the lumbar cord.

The identity of the pathological process in the cord and in the other tissues seems to show that they are not related as cause and effect, but form parts of some general disturbance of nutrition.

Tumour of Pons Varolii. BY F. T. MILES, M.D. (*Archives of Medicine*, Oct. 1881.)—Female, ætat. 17. Well-marked cross paralysis. Complete paralysis of left side of face (involving the orbicularis and frontalis), and of the right arm; incomplete paralysis of right leg. Sensation abolished in left half of face, conjunctiva, and nostril; much impaired in the right upper arm; diminished in the right leg. Hearing of left ear impaired. Paralysis of all the muscles of the left eye; inflamation of left cornea.

Post-mortem.—Externally the pons appeared enlarged and nodulated, the left side especially; the peduncles, especially the left, showing the same appearance. On section it was seen there was a growth occupying the left half of the pons, pushing the raphe to the right. J. A. ORMEROD.

Ball on Erotomania (*L'Encéphale*, 1883, No. 2).—Professor Ball narrates in detail the history of a typical case of erotomania *proper*, as distinguished from the mental disturbances peripheral in origin—nymphomania and satyriasis. He recognises the usually accepted views of its essentially centric origin; the chaste and honourable nature of the passion aroused, and frequent association with the nobler qualities of the mind; its affinity to the forms of "Religious Melancholia"; and the tendency of the erotomaniac to pass through a later stage of melancholia, with delusions of persecution, into hopeless dementia.

Luys on the Cerebral Cortex (*L'Encéphale*, 1883, No. 2).—Professor Luys gives the results of his researches into the structure of the cerebral cortex carried on by means of micro-photographic processes similar to what he formerly described in the 'Archives of Physiology' for 1875. He regards his method as likely to reconcile or explain away the discrepant views held by different observers by the substitution of an exact scientific method of delineation in place of the more fallacious individual judgment. On most points of structural detail Prof. Luys is in harmony with the majority of authorities upon the constitution of the cortex; but he differs in one important consideration from all previous writers, for he wholly ignores the usually accepted division of the cortex into five or six laminated zones of nerve-cells, affirming that we might as readily admit fifty or sixty layers into its composition! Whether or not the process advocated by Prof. Luys is likely to explode the usually accepted ideas of cortical lamination based upon the graphic descriptions of Meynert, Lockhart, Clarke and others; or whether the micro-photographic process in its present stage of development can be relied upon for the elimination of the "personal error" due to subjective bias, may be best decided by an examination of the micro-photograph accompanying Prof. Luys' memoir.

Pozzi on Sclerosis of the Cerebral Convolutions (*L'Encéphale*, 1883, No. 2).—A peculiar and hitherto undescribed form of disseminated sclerosis of the cerebral convolutions resulting in atrophy is the theme of this article. The clinical history of the case is unfortunately somewhat defective; but the coarse appearance of the lesions, topography, and histological examination are detailed *in extenso* with illustrations. The worm-eaten aspect of the cortex-to which the author draws attention, was associated with sclerosed prominent patches identical with the form of hypertrophic cirrhosis described by Bourneville and Brückner (Encéphalite tuberculeuse). In the hypertrophied patches the neuroglia gave evidence, by its density, reactions to pigments and histological features, of being the site of a chronic inflammatory action probably originating in this tissue: the nerve-cells were very sparsely scattered, the pyramidal cells very few in number, and the large "motor cells" almost entirely absent. The neuroglia has likewise infiltrated with a colloid material. In the atrophied and granular patches the condition was similar, with the addition of a still further condensation of the neuroglia and great paucity of the cellular elements. His arguments strongly favour the assumption that the atrophic sclerosis is but a later stage to which the hypertrophied nodules eventually succumb. W. BEVAN LEWIS.

Bandelette de l'Uncus de l'Hippocampe (*Archives Italiennes de Biologie.* Tome 11, Fasc. 11).—Professor Giacomini draws attention to certain peculiarities observed by him in the brain of man and the higher Quadrumana, a description of which he has failed to discover in the writings of Krause, Henle, Schwalbe, Retzius, and other anatomists, and which concerns a certain appendage to the uncus hippocampi. Upon careful exposure of the hooked termination of the gyrus hippocampi, he invariably finds (in the fresh brain), the uncus traversed by a grey gelatinous band, measuring 1 to 1·2 mm. across. Emerging from the depths of the hippocampal fissure, this band runs more or less obliquely across the inner and upper aspect of the uncus, measuring from its apparent origin, to its termination, from 2 to 3 centimeters. This structure, which is found constant in man, when traced into the fissure of the hippocampus is found to bend suddenly backwards, to be immediately continuous with the fascia dentata. By means of a minute enquiry into the structure of the uncus, and its superficial band, Giacomini shows that both are identical in constitution with that of the cornu Ammonis and its fascia dentata, but owing to the sudden change in direction taken by the superficial band of the uncus, transverse sections of this part afford appearances which are at first sight most deceptive, and have not been fully appreciated by former observers. If, however, sections are taken parallel to the axis of the uncus, this superficial band is found to be constituted of a superficial and deep layer of granules continuous with each other at one extremity, but separated at their other end, and receiving betwixt them at this latter site a series of pyramidal cells, which are but an extension from the pyramidal layer characteristic of the cornu Ammonis. We have therefore in these sections through the uncus a repetition of the layers met with in ordinary transverse sections through Ammon's horn, but beyond and outside the four layers proper to the horn we have as constituents of the superficial band the following:—

5. A layer of pia mater and blood-vessels separating uncus from its band.
6. Internal molecular layer.
7. Internal granule layer.
8. Intragranular layer, containing pyramidal cells.
9. External granule layer.
10. External molecular layer.

The oblique direction taken by the superficial band of the uncus sufficiently explains many of the anomalous appearances presented by transverse sections some distance in front of the tip of the uncus. The fascia dentata is larger and more voluminous in apes, its free border smooth, and the peculiarities presented by its anterior end are described as they occur in macacus and cercopitheous. In cercopitheous the dentate fascia is disposed as in man, but numerous and closely-packed *globose* cells, like those of the peripheral ganglia take the place of the pyramidal elements in the intragranular stratum. In lower mammals the dentate fascia is more voluminous, especially at its posterior end; and in the dog and sheep its free border is smooth, and resembles closely a convolution. In these animals it terminates at the most anterior part of the gynus hippocampi with a very gentle—scarcely perceptible—bend, over a poorly developed uncus; in fact, the superficial band may here be said to be absent. Tracing the developmental history of the region in man, and the higher mammalia, Giacomini concludes that the ending of the superficial band in the uncus is a characteristic feature of the brain of man and the higher quadrumana, corresponding to the great development in them of the pes hippocampi and its anterior bend or uncus.

Histologie de la Région psycho-motrice chez le Nouveau né. Par Magalhães Lemos. (*From an Inaugural Dissertation.*) —The author describes the cortex of the paracentral lobule in the new-born infant as being 3 mm. in depth and as divisible macroscopically into seven layers alternately more or less translucent and microscopically constituted as follows:—

1st layer. An interstitial basis substance, containing cells small in size, but possessing relatively large nuclei.

2nd layer is a well-defined stratum of nucleated cells, larger than those of the preceding.

3rd layer. The cells of this layer are more scattered and less in size than those of the second layer, whilst spherical nuclei, strongly coloured by carmine, are also found here.

4th layer. This layer is formed by a closer approximation of similar cells; the external limit of this layer is not so clearly defined.

6th layer. This layer is characterised by cells of pyramidal form, and of larger dimensions, amongst which are elements similar in structure and distribution to the the preceding layer.

7th layer. Presents no feature peculiar to itself.

It is to the existence and peculiar constitution of the large elements of the sixth layer that the author calls attention. These cells measure ·030μ to ·040μ long, by ·09μ to ·015μ in breath; some scarcely attain this size, but others reach the dimensions of ·65μ by ·020μ. Amongst these are scattered round cells having a diameter of ·020μ to 030μ.[1] The pyramidal cells stain strongly with carmine, and enclose a nucleus ovoid or elliptic in form which in the larger cells may measure ·009μ to 015μ. in long diameter. Of the branches given off by these cells, that from the apex is the most developed; it describes a centrifugal course, and may be followed to a distance of two to five times the length of the cell. Besides the other branches arising from the base of the pyramid, there is a prolongation from the centre of the cell which takes a centripetal direction (towards the medulla). The sixth layer is especially the site of these large pyramidal cells, although they may exceptionally be traced as smaller elements into the fifth or even the fourth layer. They are not disposed in regular series, as in the cornu Ammonis, but as an irregular interrupted formation, forming more or less circumscribed cell aggregations. An examination of the cortex of the internal aspect of the first frontal gyrus reveals the fact that although it is in direct continuation of the paracentral lobule the histological constitution marks it off as a region entirely distinct from the latter. The author briefly discusses the histological structure of the cornu Ammonis in the brain of the new-born infant, and declines to subscribe to the opinion of Prof. Betz, who has stated that "Ammon's horn (in the new-born infant) is already formed, possessing all its characteristic features, and that the cells are as distinctly seen as in the adult brain."

W. W. IRELAND, M.D.

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[1] The dimensions are given as in the original memoir; yet the signification of μ being usually read as *micromillimetre* (·001 mm.), there is evidently an error here, and the measurements must be taken as *fractions of a millimetre*. This explanation would accord with already ascertained measurements of the large ganglionic cells of this region in the adult.

BRAIN.

OCTOBER, 1883.

Original Articles.

ON CHRONIC ATROPHIC SPINAL PARALYSIS IN CHILDREN.

BY A. HUGHES BENNETT, M.D.,

Physician to the Hospital for Epilepsy and Paralysis, Regent's Park; and Assistant Physician to the Westminster Hospital.

In the number of 'Brain' for April 1883, English readers were gratified by an article from Professor Erb of Heidelberg, entitled "On Chronic Atrophic Spinal Paralysis in the Child." The views of so distinguished a neurologist will have been received with the attention and respect which they merit. Having had some experience in this interesting clinical condition, I venture to make a few observations on the subject, with special reference to this paper.

In the first place, I would remark that, throughout what is to follow, the expression "Atrophic Spinal Paralysis" is adopted only because it is the terminology employed by the German author, and not because I consider the name a suitable one. In my opinion it embraces too much, applying, as it does, to a somewhat extensive group of diseases, and might include any form of paralysis accompanied with muscular wasting, due to a lesion of the spinal cord, such, for example, as progressive muscular atrophy. A new and more special nomenclature is therefore required clearly to differentiate this particular state from other allied but distinct clinical conditions. In the present paper I understand by the term atrophic spinal paralysis that disease which is more commonly called "chronic anterior poliomyelitis," "chronic infantile paralysis," or the "paralysie spinale antérieure subaiguë" of Duchenne.

The object of Professor Erb's remarks is to show that chronic anterior poliomyelitis, although by no means uncommon in the adult, is (unlike its acute prototype) almost an unknown disease in young children, and he records in detail the facts of a case which has occurred in his practice. In referring to the literature of the subject, he points out that, with the exception of an instance in a girl of fifteen years old described by Dr. Ross, there is no example of the kind hitherto published, except one referred to by myself in my work on electro-diagnosis.[1]

He, however, questions the correctness of the diagnosis in this particular instance, and considers the case which he puts forward as "unique," regarding the youth of the patient, namely six years of age. That I at the time recognised the true nature of the affection in a child two years old is evidenced by the fact that the observation is headed "poliomyelitis anterior chronica." I am willing, however, to admit that the details of the case were not given with sufficient extension to establish the diagnosis, to the satisfaction of others, as my object was not to prove the rarity of the affection, but only to instance certain physical electrical phenomena in an individual muscle of the patient in question. Further, I did not at the time fully recognise the importance of the case itself as being of such exceptional occurrence, as I believed that I had seen other examples of a similar nature. Fortunately I have retained the full notes of this particular patient; and as so experienced an observer as Professor Erb pronounces an instance of the disease to be of such great interest, I make no excuse for recording the case shortly in detail, which will, I think, establish my original diagnosis to have been correct. I shall also add the records of others which have come under my observation and which I believe to belong to the same category. The facts of the disputed case are as follows.

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[1] 'A Practical Treatise on Electro-diagnosis in Diseases of the Nervous System,' p. 121; H. K. Lewis, London, 1882.

I. *Case of Chronic Atrophic Spinal Paralysis in a Child two years of age.*

Charles S., æt. 2, was brought to the Westminster Hospital on the 2nd of November, 1880. The patient was stated to have been in good health till he was eighteen months of age. When a year old he began to walk, and soon after he was able to run about like other children, and this continued till he was one and a half years old. About this time he suffered from whooping-cough and measles, from which, however, he rapidly recovered. A month afterwards it was for the first time noticed that there was slight weakness and unsteadiness in his gait, and that he had a tendency to fall down when he ran. These symptoms, scarcely noticeable at first, gradually increased in severity, and have continued to do so up to the present time. Latterly it has been observed that the patient has been awkward with his hands, but the parents have noticed no other peculiarity of the upper extremities. The general health of the child has been uniformly good, and, with the above exceptions, he has never had a day's illness in his life.

At present the appearance and general condition of the patient are healthy in every respect. He is fat and well-nourished; there is no spinal curvature; and all his functions, except those to be described, are normal. When placed on his legs, he can barely support the weight of the body, even with assistance. The limbs are weak and shaky; attempts to walk are very uncertain and imperfect, and only clumsily effected when the patient is partially supported. When sitting on his mother's knee, the lower limbs are moved about, but not with the vigour of health. The movements of the arms are active, but the actions of the hands and fingers are clumsy, and the child picks up a penny from the table in an awkward manner. Owing to his youth, it is difficult to determine exactly the precise deficiency of these movements. For the same reason the sensibility of the skin cannot be accurately ascertained; but, so far as can be made out by touching the child unawares, or pricking him when he is not looking, and otherwise testing its condition, it seems normal, as his attention is attracted by the slightest touch, and he is irritated by the gentlest prick or pinch. The plantar reflexes are well developed; the cremaster phenomenon distinctly marked; and the knee-jerk present on both sides, but not exaggerated. The limbs and joints are flaccid, and there is no trace of rigidity. The appearance of the skin of the upper limbs is normal. Here, as well as all over the body, there is a great development of fat; but, in spite of this, distinct atrophy of the extensors of the forearms and the small muscles of the hands on both sides is visible. There is no apparent loss of bulk in the muscles of the lower extremities, but the skin, especially on the left side, is cold and cyanotic. There is no affection of the bladder or rectum, nor are there any cerebral symptoms.

Electrical reactions.—As far as can be determined in a restless fretful child, they are as follows. The *faradic* current, when applied to the trunks of the ulnar nerves, causes on both sides normal muscular contractions. The reactions to both median nerves are considerably diminished, but not lost to a strong current. The greatest power of the battery fails to produce any effect on either musculo-spiral nerve, when irritated in the upper arm above the elbow-joint. A moderate current applied directly to the flexors of the forearms causes apparently healthy contractions; but, even when very strong, to the extensors it fails to induce any result except slight action of the extensor indicis on the right, and feeble contraction of the extensor ulnaris on the left side. To the other extensors the most powerful currents are followed by negative results. The *galvanic* current induces apparently normal contractions to the trunks of both ulnar and median nerves. A very strong current to the musculo-spiral nerves causes extension of the wrist and fingers, but slowly and imperfectly, K C C > A C C, but the latter increased. The galvanic current applied directly to the flexors of the forearms induces vigorous contractions, and the A C C is distinctly increased. To the extensor indicis of the right side the response is diminished, A C C > K C C, the contraction being slow and tonic. The same is observed in the extensor ulnaris of the left side. To the other extensors of the wrist and fingers and the small muscles of the hand, the strongest galvanic current fails to induce any contractions. Electrical investigation of the nerve-trunks and muscles of the lower limbs proves them to be normal, as also are those of the trunk and upper arms. Galvanometric observations show that the difference in the muscular contractions is not due to any special abnormal resistance.

May 1st, 1883.—The patient was seen on this date, that is, nearly two years and a half after the foregoing report was made. He was found in perfect health and without a trace of paralysis, or other abnormal symptoms. The mother states that for six months he was kept in bed and treated with galvanism to the spine, that improvement gradually ensued, and that at the end of this time he had completely recovered.

Although the record of the facts of this case is admittedly not perfect, as it is almost impossible to conduct delicate sensory and electrical investigations in a child only two years of age, they appear to me to furnish sufficient evidence as to the nature of the lesion, namely a chronic anterior poliomyelitis, probably limited to the lower cervical and upper dorsal regions of the cord. The disease began insidiously, and progressed slowly; it was attended with weakness and wasting of the upper extremities, with no apparent illness of any kind, with no alteration in the general health or nutrition, and with no abnormal sensory symptoms or disturbance of the bladder or rectum. It was accompanied with positive electrical abnormalities in the nerve-trunks and muscles of the upper limbs, as already described. The lower extremities, although paretic, were not wasted; they possessed their sensibility and reflex actions intact, and the electrical reactions were normal. The weakness of the legs was therefore probably not the result of disease of the grey matter of the corresponding segments of the cord, but due to interruption of the voluntary impulses by the disease at a higher level. This is contrary to the usual ascending nature of the disease; but instances are not wanting in the adult, and referred to more especially by Duchenne, where the affection was descending in nature, beginning in the upper and spreading to the lower extremities. The exact limits of the poliomyelitis could not, under the circumstances, be fully determined, owing to the difficult and uncertain task of investigating so young a subject. Although there was no actual confirmation of the diagnosis in this case, the patient having recovered, the above seems the most plausible explanation of the facts, namely that it is an example of limited chronic atrophic spinal paralysis.

The following is also a case which has recently come under my observation.

II. *Case of Chronic Atrophic Spinal Paralysis in a Child one and a half years old.*

A. C., æt. 1½ years, male, was seen by me, in consultation with Dr. Bristowe, on the 11th of October, 1882. The patient is the child of a medical man, from whom a very accurate and trustworthy account was obtained. From his birth he has been in uniformly good health, and nothing in his condition attracted attention till he was about eight or ten months old. It was then observed that although he moved his limbs freely, they were weak, and the legs did not in any way serve to support him when he was placed upright. The entire body, moreover, was flaccid and limp, so that he could not maintain a sitting posture, and he fell about "all of a heap." This condition has slowly and gradually increased till the present time, and the child has never been able to walk, or even stand or sit upright. There has been no fever nor illness of any kind, no pain nor other abnormal symptoms, and the functions and general nutrition of the body have been uninterruptedly good.

At present, the patient is a plump, rosy, healthy-looking child. The intelligence, as far as can be ascertained, is normal. The movements of the upper extremities seem to be naturally performed, but it is very difficult to estimate any delicate changes in so young a child. The muscles of the neck and trunk are evidently weak. The head has a tendency to fall in various directions, and the patient prefers to keep it supported by his nurse's arm. The child cannot sit up by himself, but falls down "all of a heap," generally forwards, and when placed on his face or back, he cannot raise himself into an upright posture. The lower limbs are voluntarily moved about, but not with the vigour of health, and the patient cannot support the weight of his body on his legs, which sink beneath the effort.

The plantar reflexes are totally absent, and there is no trace of the knee-jerk phenomenon on either side. There is no curvature of the spine, but the back is much rounded, evidently from the habit which the child has of lying in a forward position. As far as can be determined, the sensibility is not impaired. There are no definite vaso-motor changes, nor is there any visible wasting of the limbs; but it must be remarked that there is a large deposition of subcutaneous fat, which may obscure the condition. There is no affection of the rectum or bladder.

Electrical reactions.—As far as the youth of the patient permits these are ascertained to be as follows. The most powerful faradic and galvanic currents (40 Leclanché cells), when applied to any of the nerve-trunks of the body, fail to induce any muscular contractions. The strongest faradic current also has no effect when applied directly to any of the muscles of the limbs. A moderate galvanic current to the muscles is followed by negative results, but a strong one (from 40 Leclanché cells) induces feeble contractions of a slow and tonic character A C C = K C C. No muscle is specially more excitable than another. Those of the trunk are, from difficulty of manipulation, not examined. Galvanometric experiment shows no abnormal resistance.

For six months the patient was treated with both central and local galvanisation. When seen on the 24th of April, 1883, he continues in robust general health. He appears intelligent, and although he cannot speak, he tries to do so. He has grown considerably, and has much improved in strength. He can now sit up by himself and play, and there is no tendency for the head or body to fall. The movements of the upper limbs are fair, but it is difficult to estimate their exact condition. The lower extremities move about freely, but are still weak, and the patient cannot support the weight of his body. The plantar and knee-phenomena are absent, but there is distinct evidence of the cremaster reflex. The sensibility is normal. There is now marked wasting of the muscles of the legs and thighs, and those of the arms are very soft and flabby. The skin of the feet is cold and blue. Otherwise the condition is the same as before. The electrical reactions are changed only in this respect, that now a strong galvanic current, when applied directly to the muscles of the legs, indicates that A C C > K C C, the former being twice as vigorous as the latter.

In my judgment there can be little doubt that this also is a case of chronic poliomyelitis anterior. Whether the child has a normally developed intellect, is a question almost impossible to settle, owing to his youth, but the parents declare that they have no reason to suppose the contrary. Assuming this to be the case, this patient presents all the typical phenomena of the disease under consideration, and that of a chronic nature, as the symptoms arose in so insidious a manner that it is impossible to fix the date of their commencement. Moreover, for at least six or eight months, they gradually increased in severity, and only after that time seemed to be arrested in their further progress.

The following case is that of a girl of 14 years of age, who, however, was ill-developed for her years, and had not reached the age of puberty, so that she may be considered as a child.

III. *Case of Chronic Atrophic Spinal Paralysis in a Girl fourteen years of age.*

Augusta B., æt. 14, was first seen at the Westminster Hospital on the 10th of October, 1882. The patient is stated to have been in good health till about five months ago. Without any illness or apparent cause, she then for the first time noticed a slight weakness of her legs. This slowly increased, so that in two months she had much difficulty in walking. For the last eight or ten weeks she has been unable to stand on her feet, but has been compelled to crawl about on her hands and knees. The general health has throughout been good, there has been no pain nor distress of any kind, and the entire body above the lower extremities is perfectly normal.

At present the patient is a healthy-looking girl. She has no complaint of any kind except inability to use her legs. There is no abnormal condition of the spine, and no rectal nor vesical disturbance. The patient cannot stand or walk unsupported, but with a little assistance she can manage to shuffle across the ward. Her gait is very feeble, the legs sink under the weight of the body, the knees are crossed over one another, and the patient treads on the outside of her feet. When lying on her back in bed, the movements of the thigh on the trunk can be performed, but they are obviously defective. The actions of the knee-joints are slow, weak, and jerky. The movements of the feet and toes are very weak and imperfect on both sides. When at rest, the knees point inwards, as well as the feet and toes, the last being in a condition of equino-varus. There is no trace of rigidity of the muscles or joints, which, on the contrary, are soft and flaccid. The plantar reflexes and knee-jerk are absent on both sides. The sensibility is everywhere normal, and there is no pain in the back or limbs. The muscles of the thighs are very thin and spare, and those of the calves and peroneal regions are markedly atrophied on both sides. The skin of the legs is cold and cyanotic. The remainder of the body is normal.

Electrical reactions.—Above the umbilicus these are normal. Below this level all the nerve-trunks respond very feebly to the strongest *faradic* currents, and to those below the knee there is no response whatever. A strong *galvanic* current to all the nerve-trunks of the lower limbs causes contractions, but these are imperfect and below the normal vigour. When the current is applied directly to the muscles of the thighs, the contractions are vigorous, K C C > A C C, but the latter is increased. To the flexors and extensors of the feet and toes the response to galvanism is diminished in quantity, and A C C = K C C, the contractions being slow and tonic.

May 20th, 1883.—The patient was seen to-day. She has been at home for some months without any treatment. She is in much the same state as regards her powers of locomotion and the other conditions of her limbs as that already described when she entered the hospital. There has been no opportunity of testing the electrical reactions.

In this patient there can be little doubt that we have a typical clinical picture of the train of symptoms resulting from chronic disease of the anterior cornua of the cord.

In addition to the three cases above detailed, I am convinced that I have seen others in children of a similar nature, although I am not at present in a position to give an account of their condition in a sufficiently extended manner to establish the diagnosis, and, moreover, a description of them would only be a repetition of the same facts. I have also met with several cases of a more local paralysis which was probably due to disease of the spinal grey matter, which, however, could not be detailed here, as, in the absence of a post-mortem examination, there always remains a doubt as to the central origin of the disease. At this moment I have under observation a child of three years old suffering from weakness of one arm, with wasting of the muscles, and no loss of sensibility, which came on gradually in a progressive manner, and which to electrical investigation presents all the conditions already enumerated to both nerve and muscle. I regard this as a limited poliomyelitis, as the paralysis occupies the entire limb, and not, as in peripheral palsy, a single nerve or the muscles which it supplies. There is not *à priori* any reason to suppose that this disease should be peculiar to adults, and specially absent from young subjects. Is it not rather more probable that hitherto it has been overlooked or confounded with other affections? As the more precise methods of modern investigation become more widely spread, as electrical and other physical tests are more universally employed in the diagnosis of nervous lesions, it will doubtless transpire that the disease under discussion is not so rare as is at present supposed.

To arrive at a correct conclusion, as to the presence of atrophic spinal paralysis, is only a question of accurate diagnosis; and to the neurologist who has been initiated into all the modern methods of investigation, this is as a rule not a difficult task. For this purpose he must not depend on any special or characteristic symptom alone, but must fully consider all the circumstances of the case and bring to bear on the subject all the critical enquiry and armamenta of physical research which he possesses. Poliomyelitis, in its chronic form, if we except lead palsy, which it is improbable would occur in a very young child, is not likely to be taken for any other spinal disorder except progressive muscular atrophy, which affection, according to Duchenne, is not uncommon in children. In both these diseases the anterior cornua are degenerated, though at present we have no reliable pathological distinction between the two. But the clinical sequence of symptoms in each case is so different as to warrant us in regarding them as separate affections. In chronic atrophic paralysis the parts are attacked "en masse," while in progressive muscular atrophy they are affected piecemeal. In the former the paralysis appears first, and the atrophy of the muscle follows; while in the latter there is no real palsy, but the weakness is in proportion to the loss of muscular substance. In the first, the nerves, both at their trunks and peripheral extremities, are degenerated; in the last they remain for long intact, hence the marked difference in the electrical reactions. Finally, in poliomyelitis the reflexes are abolished; while they are present till a late stage in progressive muscular atrophy. Why degeneration of the anterior cornua should, without perceptible anatomical difference, cause two diseases unlike one another, we do not yet know, and it remains for future enquirers to determine the special anatomical distinction in such case. The fact remains that what appears to be the same morbid process is followed by two clinical conditions, not only unlike in symptoms, but dissimilar in progress and termination.

It is certain that, in the distinction between atrophic spinal paralysis and progressive muscular atrophy, the most important agent which we possess is the electrical test. Not only is this a means of determining the difference between these two diseases, but it is the most rapid and certain method of distinguishing poliomyelitis from many other doubtful and obscure paralyses. In children there may be many forms of weakness due to a variety of causes, such as brain disease, compression of the cord, simple emaciation, &c., which are often confounded together, and which it may sometimes be difficult or almost impossible to differentiate. The electrical test alone gives us the most reliable and definite information in distinguishing these and other conditions from that paralysis which is due to degeneration of the anterior cornua of the cord.

When the poliomyelitis is limited, and the paralysis resulting therefrom is confined to a limb or part of a limb, the diagnosis is sometimes very difficult. It is then far from easy to determine whether the symptoms are due to a cord or to a peripheral nerve lesion. The same general rules already referred to must be considered. In addition, the fact of the paralysis being confined to the trunk of a single nerve, or distributed more profusely, the condition of the sensibility and the history of the case, will further assist us in the solution of this delicate problem.

The electrical reactions in the three cases above detailed were definite and characteristic, and accorded in every particular with those found in acute infantile paralysis, and in the chronic poliomyelitis of adults. There was great diminution or total loss of response to both the faradic and galvanic stimuli when applied to the nerve-trunks of the affected parts. The muscles, when directly irritated with the induced current, also failed to respond, but galvanism to these caused contractions more or less vigorous, of a slow and tonic character, with the polar phenomena altered or reversed—in short, the typical "reaction of degeneration." In all the cases of poliomyelitis which have come under my notice, this condition, or a slight modification of it, has existed, namely alteration in the excitability of both nerve and muscle. In Professor Erb's case, the conditions were somewhat different. Here, when the nerve-trunks were stimulated with both currents, there ensued practically normal contractions, and the same took place when the muscles were directly irritated with faradism. With galvanism, on the other hand, the muscles displayed tonic contractions with inverted polar reactions, the whole constituting what has been termed "the middle form of the reaction of degeneration." I have not met with this condition in poliomyelitis, as in all the cases which I have investigated the nerves were distinctly altered. If the last condition was universal, the test would serve to distinguish this disease from progressive muscular atrophy, in which the nerves are generally normal. The case under notice cannot have been an example of this affection, as it rapidly recovered, and the reflexes were absent. At the same time I consider that the electrical reactions described were not those usually met with in either acute or chronic poliomyelitis, but rather those seen in progressive muscular atrophy. A possible explanation of the unusual phenomena in this instance may be that here the disease was ill-developed or slight, as evidenced by its subsequent rapid and complete recovery. The lesion of the nerves was sufficient to cause certain peculiarities in the electrical reactions (the partial reaction of degeneration and the sluggish contractions), but not to render them altogether insensitive to stimulation, as indicated by muscular contraction following indirect irritation. These facts are of prognostic value, as both Erb and Kast have suggested, pointing to a mild progress of the disease and a favourable termination.

An imperfect development of the disease may also explain the presence of the peculiar phenomena described in Professor Erb's case, following both direct and indirect excitation by the two currents, namely the great tonicity of the contractions. I have seen this in certain cases of muscle-degeneration, but never, as far as I can recollect, in poliomyelitis. In this, as in other deviations from the usual type, we are not yet aware of its exact clinical significance. I have occasionally observed another variety of altered reaction,[1] the explanation of which is equally obscure. In certain diseased conditions of muscle, a rapidly interrupted galvanic current causes a sudden and vigorous contraction, immediately followed by relaxation, although the current continues to flow. In a healthy muscle this would of course be accompanied with tetanus during the application of the electrode, whereas in the above instance the contraction is momentary. This condition I have seen in peripheral paralysis,[2] lead poisoning, &c. It would seem as if in certain forms of muscular degeneration without complete disorganisation, the shock of the interrupted galvanism causes contraction, but that the muscle, owing to disease, has lost its tonicity and capacity to maintain a tetanic spasm.

Those modified electrical reactions, those deviations from the usual types, of which we are at present ignorant of the full significance, will doubtless, as our experience extends, prove valuable agents in the diagnosis and prognosis of disease.

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[1] 'Electro-diagnosis,' p. 78.

[2] *Loc. cit.* p. 139.

ON INFANTILE SPASMODIC PARALYSIS.

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THE cases which I am about to describe in this paper possess one striking and characteristic feature. Whatever may be their pathology—and on this point there is ample scope for discussion—they are all linked together by one clinical bond. I refer to the condition of the lower extremities.

Although in many cases other symptoms exist, which when rightly considered assist greatly in the interpretation of the clinical phenomena, a peculiar attitude of the legs and a characteristic gait are common to all.

The lower extremities are more or less flexed at the hip and knee-joints, the thighs are rigid and adducted, the knees in contact, the legs inclined outwards, and the heels often drawn up from the ground by the contraction of the gastrocnemii. Briefly, this condition is one of spastic paraplegia. But in the child, adductor spasm seems to prevail; whereas in the adult extensor spasm is commonly observed.

The cases which have come under my own observation may be divided broadly, and for the sake of convenience, into two groups.

1. Cases of spastic paraplegia, in which certain nutritive changes are present in one upper extremity.
2. Cases in which the lower limbs alone are affected.

It must be mentioned that several instances of all four extremities being involved are also on record, but this was not present in any of my own cases. I shall, however, take the opportunity, when discussing the nature of the disease, to allude to this condition.

Of the first group I have but two cases to relate. In one[1] the patient was a boy, aged 13, whose family are free from all neurotic taint. He has been subject to fits, occurring at intervals of from one to five months, or more, since he was a year old.

When a child, three years of age, he had a fall, since which (according to the friends' statement) he has been paralysed in both legs and right arm. The fits are preceded by irritability of temper, and by a distinct aura, consisting of a "nasty taste" in the mouth.

Movements, with trembling, then begin in the right hand, and rapidly extend over the rest of the body.

During the attacks he bites his tongue and foams at the mouth. Afterwards, he is drowsy, dull, and sometimes so weak that he is confined to bed for four or five days.

With the exception of chicken-pox, and the presence of worms when he is constipated, the patient has suffered from no other complaint.

The boy was admitted into St. Thomas's Hospital, under Dr. Bristowe, on account of a severe epileptic attack.

When first seen, he was strongly convulsed, and there were clonic spasms of the hands, especially the right. The upper extremities were far more affected than the lower, which, with the exception of a few slight twitches, were motionless. The muscles of the face and eyeballs were strongly convulsed; the pupils constantly varied in size. The patient was quite unconscious. The patellar reflex was said to be entirely absent, although there was marked ankle-clonus.

On subsequent examination, the patient was found to be a fairly intelligent boy. Although his friends consider him somewhat deficient mentally, he reads, counts, and remembers well. His expression is rather vacant, but there is certainly no notable intellectual deficiency. The pupils are equal and act well, both to accommodation and to light. There seems to be slight lateral nystagmus. The tongue is protruded straight, and there is no facial paralysis. The right upper extremity is much smaller than its fellow, the ulna being an inch shorter than the bone on the opposite side. The muscles are considerably wasted, and Dr. Kilner, who examined them electrically, said they all present the reaction of degeneration. The fingers are kept apart, hyper-extended at the metacarpo-phalangeal joints, and semiflexed at the other joints. This condition is especially marked in the index and middle fingers. There is very pronounced loss of power both of the arm and hand. The tendon-reflexes are much more evident than on the left side.

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[1] "An Anomalous Case of Infantile Hemiplegia;" 'British Medical Journal,' Feb. 18th, 1882.

The left upper extremity is in every respect natural.

The legs are rigid, and there is well-marked talipes equinus on both sides. The right lower extremity is decidedly stiffer than the other, and the contraction of the gastrocnemius on the left side is not so great as on the right, since he can, although with some little difficulty, bring the left heel to the ground. The patellar reflex is exaggerated, but there is no appreciable difference between the two limbs in this respect. Ankle-clonus was usually obtainable on both sides to nearly an equal extent, but on the last occasion when it was tried it existed on the right side only. Occasionally it was impossible to provoke it, in consequence of the great rigidity of the calf-muscles.

The lower limbs are not atrophied, the electrical reactions of the muscles are normal, and there is no loss of power.

The plantar reflex is present on both sides, but it is difficult to say whether it is exaggerated or not.

The abdominal reflex exists on the left side only. The right cremasteric reflex cannot be obtained in the usual way. On stimulating the inner aspect of the left thigh, both testicles are simultaneously retracted.[1]

Sensation is everywhere intact, and there is no loss of control over bladder or rectum. There is no atrophy of the thoracic wall or pelvis on either side.

But the most striking feature in this patient is his gait. When asked to walk, he supports himself for a moment by leaning against some object. It is evident, indeed, that, although not lacking the muscular force, he is unable to maintain his equilibrium, when upright, without extraneous aid.

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[1] I had the opportunity of examining this boy since the above was written. The plantar reflexes are decidedly exaggerated, the right being the most marked. On stimulating the inner side of the right thigh, I succeeded in obtaining a slight reflex movement of the corresponding testicle.

As he stands, he is seen to rest on the balls of the toes, the heels are drawn up from the ground, the hip and knee-joints are slightly flexed, the thighs are adducted, the knees almost in contact, the legs incline outwards, and the feet are much inverted. During progression, he brings each lower limb forwards by describing an arc with the convexity outwards. At the same time the head and shoulders are thrown back, the dorsal curve is greatly exaggerated, and the right arm, which is flexed on the forearm, with the corresponding hand dropped on the wrist, is brought up nearly to a level with the chin. As he walks he keeps the right upper extremity in this position; but the opposite limb is moved backwards and forwards, and, together with the trunk, which sways powerfully from side to side, serves to maintain the equilibrium during progression.

The condition I have just described depends, I believe, on a cerebral lesion, although the secondary spinal effects tend to obscure the true nature of the disease.

The fact that the epileptic attacks began in the right hand and affect the corresponding side more powerfully than the other limbs, strongly suggests that the lesion is primarily cerebral. The atrophic condition of the right upper extremity and the complete immunity of the left upper limb point to the unilateral nature of the lesion.

If the state of the lower extremities alone were considered, it might be urged with much force that the disease was spinal. It must be mentioned, however, that although both limbs are extremely rigid, the right is slightly more affected than the left. The absence of the right abdominal and cremasteric reflex is an additional argument in favour of the unilateral cerebral nature of the affection. Rosenbach and Jastrowitz have pointed out that these reflexes are lessened or abolished on the paralysed side in the case of disease of one cerebral hemisphere.

It is curious that, although the right cremasteric reflex could not be obtained by stimulation of the inner side of the thigh on the corresponding side, the phenomenon was called forth by irritation of the skin on the opposite side, and that the reflex movements were apparently synchronous. This fact seems to indicate that the motor cells in the lumbar region are more intimately connected on each side than is usually the case.

I believe that in this patient there exists some lesion or congenital defect in the motor part of the left cerebral hemisphere. This lesion, whatever may be its nature, has been followed or accompanied by change in both lateral columns, and it is highly probable that the motor cells which control the movements of the right upper extremity have undergone atrophic changes, or have been arrested in the natural course of their development.

Why, it may be asked, have both lateral columns been attacked? Cases of hemiplegic rigidity in children are not uncommon, but it is rare to find both lower limbs affected in consequence of a unilateral cerebral lesion. In a paper about to appear, I have given some cases of paraplegic rigidity in hemiplegia and have discussed their probable nature.[1]

These cases may be explained as follows. It is well known that the decussation of the bulbar pyramids is liable to great variations. The direct pyramidal tract usually bears a very small proportion in the number of its fibres to the crossed tract, but occasionally the order of things is modified or absolutely reversed.

Now, the fasciculi of Türck, which are concerned with the transmission of voluntary motor impulses, diminish in number as they pass down the cord, generally disappearing about the middle of the dorsal region.

It seems to me possible that these fibres dip into the cord and pass downwards on the same side, to enter into relation with the multipolar cells in the lumbar region. It is a matter of common knowledge that one cerebral hemisphere controls not only the movements of the opposite limbs, but to a certain extent of those on the same side.

It is quite conceivable that when the non-decussating pyramidal fibres are unusually numerous, the loss of power might be distributed between both lower limbs, and not practically limited to one leg only. Cases of this nature are not very uncommon.

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[1] "On Paraplegic Rigidity in Hemiplegia;" St. Thos. Hosp. Reports for 1882.

Charcot believes that paraplegic weakness or rigidity supervening in hemiplegia depends on a second decussation of the motor fibres in the dorsal region. In other words, he suggests that there is not only a decussation of the pyramidal tract-fibres at the medulla, but a partial and variable intercrossing of the same fibres in the spinal cord.

The atrophy of the right upper extremity is worthy of remark. It depends probably on the fact that the spinal nerve-cells which preside over the movements of the upper limbs are much less intimately connected across the cord than those which control the movements of the lower limbs.

Muscular atrophy in lateral sclerosis, whether primary or secondary, must be looked upon as exceptional. When it does occur, as in late hemiplegic rigidity and amyotrophic lateral sclerosis, the arm is most liable to be affected.

It would seem as though bilaterally associated cells were mutually protective, to a certain extent, against the extension of influences affecting their nutrition.

The next case is that of a young man, aged 19, who was admitted into St. Thomas's Hospital under Dr. Bristowe in the early part of this year. He was a poorly-nourished youth, of about average height, with decided prominence of eyeballs, but without any enlargement of the thyroid gland. The patient was born at full time and was one of twins. The birth was difficult, but no instruments were used. The other child was born dead.

According to the friends, the patient was born head first.

There was no history of fits or of any hemiplegic condition in infancy.

Both his father and mother had died of phthisis, but there was no history of nervous diseases in the family, and, as far as could be ascertained, there was no suspicion of congenital syphilis.

He began to walk when between four and five years old, and from the first walked on his toes. Later, he walked more on the soles, but about two years ago he again took to walking on his toes.

About this time he had pains in both ankles, which of late years became worse, the right hip being also affected.

On admission, he complained of weakness of legs and frontal headache.

His gait presented the same characteristics as that of the patient whose history has just been given.

As he walks, he brings one foot and leg in front of the other, and as the latter is being brought forward in its turn, the knees overlap and rub. The feet are not applied well to the ground, but rest almost altogether on the balls of the toes.

During progression, there is some lordosis, the shoulders are thrown back, and he waddles from side to side. When lying down, the legs are more or less adducted and stiff. The rigidity becomes more marked when passive movement is attempted. In the supine position the lordosis disappears, and there is no ankylosis of the hip-joints.

Both knee-jerks are very brisk, but the gastrocnemii are so contracted that ankle-clonus is only obtained occasionally, and with some difficulty.

Sensation is nowhere absent; but below the knees it seems to be a little deficient. There is no affection of bladder or rectum. The left hand is curiously deformed; the middle, ring, and little fingers are flexed at the first interphalangeal joints, and he cannot extend them. The ends of the fingers and thumb are bulbous, and the epidermis over them is very thick and hard. The nails are imperfectly developed, being small, thick and claw-like.

In fact, the terminal phalanges are much atrophied. The fingers of the left hand sometimes become more contracted, and the arm feels stiff. The hand and arm are slightly weak. Sensation is said to be a little blunted in the left arm.

The right upper extremity is quite natural, the elbow-jerk is present equally on both sides. The abdominal, cremasteric and plantar reflexes are also obtainable on both sides. The special senses are unaffected, and there is no paralysis of face or tongue. The patient is fairly intelligent, but has never given much time to school-work. All that can be said of him is that he is dull and somewhat slow of comprehension. The chest is a little deformed, the sternum being rather prominent below. There is no asymmetry of the skull, and no abnormality of palate.

The measurement beneath axilla and over shoulder is 15¾ inches on right side, and 15½ inches on left side.

The greatest circumference of right arm is 10 inches; of left, 9½ inches.

The length of right arm is 14 inches; of left, 12½ inches.

The length of each forearm is 11 inches.

The circumference of right palm is 8¼ inches; of left, 7⅜ inches.

There is no difference in the measurements of the lower extremities.

To sum up, the left arm is 1½ inch shorter than the right, and the circumference of the left hand nearly an inch less than the right.

During his stay in the hospital his sensation was several times tested and found natural, although on the first examination some deficiency was thought to exist below the knees and in the left arm. When first seen, he could only walk a few yards, as he soon staggered and fell. He improved considerably in this respect after a few weeks, but the peculiarity of the gait remained, the knees knocking together and overlapping, and the foot being swung round with a semicircular movement.

The nature of this case is even more obscure than the other, but the involvement of three limbs makes me inclined to think that the cause is primarily cerebral. The absence of fits, on the other hand, is an argument against this presumption.

The ill-developed condition of the terminal phalanges of the left hand was new to me, but I have lately seen a similar affection in a patient who is suffering from dislocation of both wrist and shoulder-joints. There is no suspicion of ataxy, but there is evidence of some degeneration in the lateral columns of the cord.

I will now pass on to the second group of cases. I take the opportunity of expressing my indebtedness to Mr. Clutton and Mr. MacKellar, of St. Thomas's Hospital, who kindly called my attention to some of these cases. I must remark too, incidentally, that patients of this class are to be found more especially in the surgical out-patient department, where not unfrequently the true nature of the disease is overlooked.

Case I.—Male, æt. 9. The father died of phthisis, and one brother in infancy, of cause unknown. There is nothing else to note in the family history. The mother states that he was not born prematurely. The labour was difficult, and instrumental aid was necessary.

He had convulsions for nearly a month after his birth, and they continued at intervals until he was a year old. No loss of power in one arm and leg was ever noticed. He has never walked properly, but began to step when about two years of age. He has never been to school, so is very backward.

The patient is a thin, pale, half-idiotic looking boy, with a large but symmetrical head, a low and retreating forehead, drooping eyelids, prominent and overlapping front teeth.

He can neither spell nor add up, he laughs and cries readily, and is very dirty in his habits, passing his evacuations under him. His condition, there is no doubt, is one of partial idiocy.

He can speak simple words and understands easy questions.

When lying down, the knees, which are slightly bent, are in contact; the thighs are adducted and nearly touch. The feet are extended, the right partially overlapping the left; the great toes are crossed, and the heels are turned outwards. He can neither bend nor extend the feet, but there is fair movement at the knee and hip-joints.

The plantar reflex is absent on both sides.

The knee-jerks are much exaggerated.

On striking the tendon the leg at once becomes extended, and remains so for a few seconds trembling, before falling back into its usual position.

Ankle-clonus can be obtained on both sides, and there is occasional knee-clonus.

The measurements of the two legs are about equal.

The abdominal, cremasteric, inter-scapular and gluteal reflexes are brisk, but not more marked on one side than another.

He can neither stand nor walk without help. When he tries to go forward he slides the foot along the ground with a semicircular movement.

The gait and the spastic condition of the lower limbs are the same as in the cases already described.

The arms are in every respect natural.

The terminal phalanges both of fingers and toes are well developed.

There is no affection of common or special sensation. The chest wall is of good shape, and both thoracic and abdominal organs appear healthy.

CASE II.—Male, æt. 8. The mother is said to have died from "asthma." The birth of the patient occurred at full time, the head presented, and delivery was quite easy.

The child was healthy up to the age of twelve months, and had cut several teeth. He then had about six fits, extending over a period of as many months. After these attacks, his friends noticed that there was something wrong with his eyes. At the age of three he had not begun to walk, and it was thought that the legs were "queer."

On examination the child is seen to be fairly nourished and slightly undersized. The forehead is prominent, the upper part of skull large and square-shaped, in marked contrast to the face, which is small. There is no asymmetry of skull and no malformation of palate.

The child is really an idiot. He is never still, always singing, is very mischievous, and peevish, and swears at the slightest provocation. He is dirty in his habits, and passes the urine and fæces under him.

The condition of the eyes is said to date from the time of the fits.

There is double internal squint, marked lateral nystagmus, and inequality of pupils, the right being the larger. The gait presents the peculiar characteristics seen in the other patients. He seems to be a little flat-footed. The knee-jerks are very brisk, but there is no ankle-clonus.

The arms are quite natural. Sensation is unimpaired. The chest is well formed.

CASE III.—Female, æt. 15. There is nothing of importance in the family history.

The patient was born at the eighth month, and was not one of twins.

There was no difficulty about the birth. She has never had fits nor anything like one-sided weakness. Practically she has never walked, although she began to make an attempt at the age of four.

The patient, who is the daughter of a clergyman, is a refined and intelligent girl. There is absolutely no mental deficiency. The legs are bent on the thighs at an angle of about 150°. The tendons of the hamstrings are very rigid.

When she tries to walk with assistance, the balls of the toes drag along the ground, the legs are strongly adducted, the knees rubbing and overlapping.

The feet are kept in a position midway between extreme flexion and extension.

The toe-nails are all extremely small and ill-developed, looking as though they had broken off short.

The patellar-tendon reflex is brisk, but the range of movement is not extensive, on account of the contraction of the hamstrings. There is double ankle-clonus. The plantar reflex appears to be absent. Sensation is nowhere impaired. The functions of the bladder and rectum are quite natural. The arms are unaffected. There is slight lateral nystagmus on looking to the extreme left. The special senses are not involved.

Case IV.—Female, æt. 3. The mother has been married four years and three months, and has had three children, of whom the patient is the eldest.

The mother had a fall when six months pregnant, and is inclined to ascribe the child's present condition to that cause. Delivery took place at the end of nine months; the head presented, but the labour was tedious and instruments were used.

At the age of ten months the little girl had a convulsive attack, affecting the left side only. A month later, the right side was similarly attacked. This is the voluntary statement of the mother. She is quite certain that one side alone was affected each time.

Nothing like hemiplegia was noticed after the fits.

The patient began to walk at the age of eight months. Dentition did not commence until she was a year old.

When two years of age she began to talk, and can say anything now. The child is of about average size, fairly intelligent, not mischievous, but rather cross. No measurements of the skull were taken. The palate is somewhat high and arched. When walking, the thighs are adducted and the legs describe small circles. The knee-jerks are well-marked and apparently exaggerated. There is no ankle-clonus. There is slight talipes equinus on both sides.

As I only saw the child once, the history is somewhat imperfect; but at the time I satisfied myself that the case fell into the same category as those above described.

In addition to the four instances I have given, two or three more of similar nature have come before me. Unfortunately I have no notes of them.

What, it may be asked, is the pathological nature of these cases? Are they of cerebral or of spinal origin?

Is the lesion, wherever it may be, of an artificial or of a congenital nature?

Dr. Ross, of Manchester, has recently recorded several instances of this condition in a recent number of 'BRAIN,' and has also given the details of a post-mortem examination.[1]

In this case the arms were also affected. On each side, in the position of the fissures of Rolando, there was a deep sulcus, which extended from the point of bifurcation of the Sylvian fissure upwards to near the longitudinal fissure. "The lateral walls of each sulcus are formed by an infolding of the grey matter of the cortex, and these walls are so closely applied to each other that there is no great cavity left between them. In depth each sulcus extends the whole thickness of the centrum semi-ovale, and opens into the roof of the corresponding lateral ventricle. The opening into the ventricle on each side is only large enough to admit the little finger, and each opening is surrounded by a ring of grey matter, having all the naked-eye appearances of the cortex. . . . With regard to the fissures and convolutions, those in the neighbourhood of the defect are radially directed towards the centre of the cavity. The ascending parietal and ascending frontal convolutions apppear to be absent on both sides."

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[1] "On the Spasmodic Paralyses of Infancy;" 'BRAIN,' No. 1, p. 477.

On microscopical examination the giant cells were absent from the walls of the cavity on each side. The cells too in the inner division of the third layer were large, round, nucleated, and devoid of processes.

The anterior pyramids of the medulla oblongata were about half the natural size, and the lateral columns of the cord were also smaller than the corresponding parts in a healthy cord. It would appear that no qualitative change was found.

Dr. Ross believes that the porencephalous defects were due to an arrest of development, and not to any destructive process.

Mr. Hutchinson has recorded a case which in many respects resembles the preceding. At his request I saw the child, and I am inclined to think that the condition is closely allied to that of the patients I have described.[1]

From a perusal of several cases of spastic paraplegia in children, it would appear that in all probability they depend on some congenital defect in the nerve-centres.

In connection with this subject it is worth while to recall the researches of Parrot and Flechsig on the development of the pyramidal tracts. The motor fibres take origin from the ganglionic cells in the cortex just below the fissure of Rolando. The process begins soon after birth, but is not completed until the end of the first year or later. The fibres gradually descend, by a kind of budding, into the crus, pons, medulla oblongata, and spinal cord.

Assuming a double porencephalous defect near the fissure of Rolando, such as Dr. Ross has described, it might happen that the downward passage of the pyramidal-tract fibres is arrested high up in its course. In that case both arms and legs would be involved. If the arrest took place at a lower level, the legs alone would be affected. This hypothesis is partly supported by the post-mortem examination given by Dr. Ross. Here the pyramidal tracts were not diseased, but simply small. It is well to bear in mind, however, that the pathological anatomy of simple spastic paraplegia in children is practically unknown.

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[1] "A second Report on a case of Double Hemiplegia of Infants, with Idiocy and a Transitory Dermatitis." 'Trans. of the Path. Soc. of London,' vol. xxxiii.

The frequent occurrence of fits, mental deficiency, and other cerebral symptoms suggests that the condition of the legs is either dependent on, or accompanied by, some lesion in the brain.

A satisfactory explanation of the two cases in the first group is somewhat difficult.

In both there was a trophic lesion in one arm, the other arm being absolutely unaffected.

Accordingly I am inclined to believe, as I have already said, that the lesion is primarily unilateral in the brain, and that the paraplegic distribution of the rigidity is due to an unusual decussation of the pyramidal-tract fibres. The cases are undoubtedly rare. I have seen no similar recorded instances.

After carefully studying my own cases, and comparing them with those of Dr. Ross and Dr. Gee,[1] I came to the following conclusions.

There is nothing special in the family history.

No condition, either during pregnancy or during birth, has any influence.

Convulsive attacks in infancy occur in nearly half the cases.

The first fit usually takes place during the first year, sometimes shortly after birth, more rarely at the second year of life. As a rule the attacks extend over a few days or even months, and then cease either completely, or recur after an interval of months or years.

The fits vary from one or two to half-a-dozen or more; sometimes they are very numerous. Unilateral attacks are rare. In one of my own cases the left side was first attacked and then the right, but on each occasion the seizure was purely one-sided. In one of Dr. Gee's cases the left side was chiefly affected, and a temporary weakness was noticed afterwards. In one case internal strabismus and nystagmus were said to follow shortly after the fits. In one case only an aura and local commencement of the fits existed.

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[1] St. Bartholomew's Hosp. Reports, vols. xiii. and xvi.

Walking is nearly always delayed. Sometimes the patients never walk. The attitude of the lower limbs is common to all the cases, and if progression be possible, the gait is the same in all.

Dentition is late. Articulation is often quite natural. Hesitation and drawling have been observed. Intelligence is natural in a large number. Mental deficiency, varying from absolute idiocy to mere dulness, is not unfrequent. Ocular symptoms occur in a considerable proportion—nearly a half. Converging strabismus is the most common; nystagmus with or without decided squint less frequent; inequality of pupils and protrusion of eyeballs rare.

The tendon reflexes are well marked, probably exaggerated, in all the cases. Ankle-clonus exists in many, but is sometimes difficult to obtain. The skull is generally natural, rarely unsymmetrical. Occasionally it resembles that of a microcephalic idiot.

Among occasional symptoms are deformity of chest, choreiform movements, cramps, arching of palate, and weakness of back.

Muscular wasting and the reaction of degeneration are unrecorded, except in one of my own cases. Sensation and the functions of the bladder and rectum are unaffected.

A word as to treatment. It is clear that drugs can be of no avail. In Case I. of the second group the tendo Achillis on each side was divided by Mr. Clutton. The result was discouraging, as might be expected from the nature of the affection.

THE PULMONARY PATHOLOGY OF GENERAL PARALYSIS.

BY J. CRICHTON-BROWNE, M.D., LL.D., F.R.S.,

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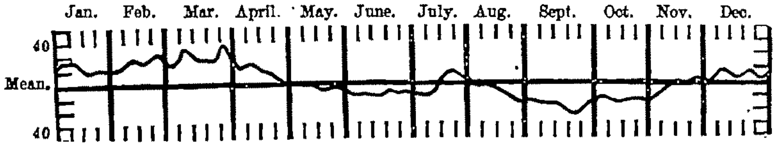
Every one who has had to make *post-mortem* examinations of the bodies of persons who have died of general paralysis of the insane must have been struck by the fact that the pathological changes in that disease are not confined to the brain and nervous system. Having their primary seat and permanent home in the cerebral cortex, these changes spread in various directions, and send forth their evil influences throughout the system, so that when the disease is far advanced, there is scarcely an organ or tissue that does not present some morbid modifications. The essential *substrata* of the diseased processes in the central nervous system must, of course, always receive a principal share of attention; but the secondary degenerations of other parts are certainly deserving of some consideration, both because they reflect light on the conditions in which they originate, and because they may alter or cut short the course of the malady to which they are themselves subsidiary.

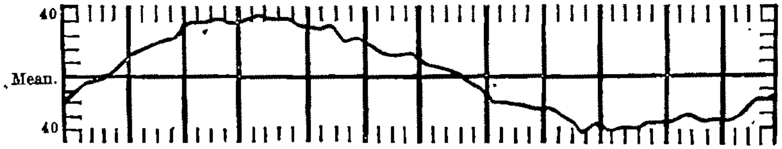
Among the secondary degenerations in general paralysis those that affect the lungs are not the least conspicuous and important. In a large majority of the cases of this disease in which autopsies are performed, coarse changes in the pulmonary structure are plainly visible, and in many instances these are of a character which indicates that they have interrupted the progress of the cerebral disorder, and cheated its victim of the dregs of life.

But although thus manifest and significant, the pulmonary changes in general paralysis have hitherto received only a passing notice. They have not been subjected to careful study or analysis, and it would be impossible to find on record to-day any definite information as to the relative frequency of their different varieties. Under these circumstances, it has seemed to me that it might be useful to sum up a few observations which I made upon these changes some years ago. The observations are somewhat superficial, and, being simply macroscopical, have not the value which they would have possessed had they been supplemented by histological investigations; but still even in their crude state they are not perhaps without interest and practical suggestiveness.

My observations were carried out in the West Riding Asylum, and were made on the bodies of 100 patients—80 males and 20 females—who died in that institution of general paralysis between April 1872 and August 1875. The average age of these patients at the time of death was 41 years and 7 months in the men, and 42 years and 5 months in the women; and the average duration of asylum treatment was 16 months and 2 weeks for the men, and 24 months and 1 week for the women. The earliest age at which death from general paralysis occurred was 24 years in a man, and 32 years in a woman; and the greatest age at which it occurred was 55 years in a man, and 57 years in a woman. The average age for the incursion of the disease is about the same for both sexes, viz. 40 years; but the advance of the malady is more rapid and violent in men than it is in women. In the 100 cases which are here reviewed, all cases of general paralysis in the West Riding Asylum that reached a fatal termination, within the period defined (April 1872 to August 1875), are included, whether death happened in the ordinary course of the disease, or in consequence of some intercurrent malady; but it is to be noted that in this group of cases intercurrent maladies did not appear until towards the natural close of the general paralysis. In every case the patient was in the fourth or last stage of the disease when death took place, and in a large number of cases that stage was so far advanced, that the intercurrent malady might be looked on rather as a mode of death than as a fatal complication. And yet evidence is not wanting that intercurrent affections, although so late in intruding in these cases, did to some extent shorten their duration. Of all such affections, those of the respiratory apparatus are the most frequent and deadly, and if we examine the distribution throughout the months of the year of the mortality in the cases which we are considering, we shall at once recognise the influence of diseases of the respiratory organs. General paralysis is essentially a nervous disease, and, like all other nervous diseases, it is most fatal in the spring months. Were the mortality which results from it, in a large number of cases, when it runs an uncomplicated course, arranged in the manner adopted by Dr. Arthur Mitchell and Mr. Buchan, in their masterly investigations into the influence of weather on health in London, it would be found that the curve which could be regarded as its constant would closely correspond with the curves of convulsions, epilepsy, whooping-cough, and other nervous diseases. In the accompanying woodcuts reproduced from a paper by Mr. Buchan, the curves for convulsions and whooping-cough are given, the straight black line in each figure being drawn to represent the mean weekly death-rate on an average of the fifty-two weeks of the year, and the figures on the margin the percentages above or below the average.

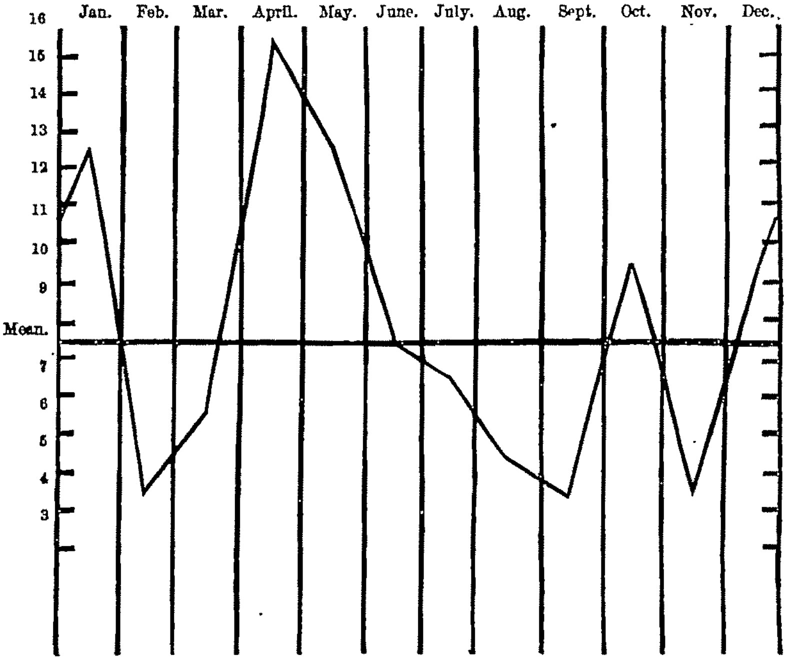


CONVULSIONS.

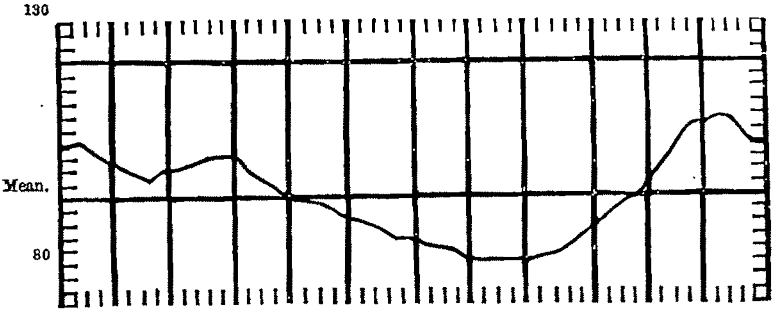


WHOOPING COUGH.

It will be gathered from these woodcuts that the maximum mortality from convulsions and whooping-cough occurs in the spring months—February, March, April, and May; and that their minimum mortality is in the autumn months—August, September, and October.



General Paralysis.



Bronchitis.

Now if the incidence of the mortality from general paralysis in the several months of the year be thus diagrammatically represented as regards the cases which we are examining, and which, although too few in number to justify any very precise conclusions, may yet supply some trustworthy indications, we shall see that this disease too has its maximum mortality in April and May, and its minimum mortality in August and September; but that its curve likewise presents a second very decided rise in December and January, in this respect resembling pneumonia and bronchitis, which have their maximum mortality in the cold months of the year.

When the curve of the mortality from general paralysis can be constructed from a number of cases so large as to obviate mere accidental variations, such as that in the November fall in the accompany chart, it will be found that it is really a compound of the curves of the mortality from nervous and respiratory diseases, and it may hence be inferred that the affections of the respiratory organs which complicate general paralysis have a certain independent influence on its course and termination.

The first point to which I directed attention in inquiring into the pulmonary pathology of general paralysis was the weight of the lungs, and the results obtained with reference to that subject are exhibited in Table I.

The mean weights of the lungs here given—26½ oz. or 756 grammes for the right, and 24 oz. or 682 grammes for the left in the male, and 18½ oz. or 526 grammes for the right, and 15½ oz. or 438 grammes for the left in the female, are so greatly in excess of all standard lung-weights, that it is obvious that they have been raised in the case of general paralytics by morbid conditions of the lungs, the influence of which is also apparent in the enormous difference between the highest and lowest weights, as shown in the Table. In order to determine to what extent the increase in the weight of the lung in general paralysis was due to pathological changes, I selected from the 100 cases all those in which the lungs were normal, or at least free from coarse and obvious pathological change, and calculated their weight separately. These cases were sixteen in number, including fourteen men and two women, and in them the lungs were practically sound, no departure from healthy structure being observed by the unaided eye beyond a slight emphysema at the margins in one

TABLE I.

SHOWING THE WEIGHT OF THE LUNGS IN ONE HUNDRED CASES OF GENERAL PARALYSIS.

—		Right Lung.						Left Lung.					
		Highest.		Lowest.		Mean.		Highest.		Lowest.		Mean.	
		Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.
Persons .	100	46·75	1326	7·50	214	25·00	710	56·50	1600	10·50	298	22·25	633
Males .	80	46·75	1326	13·00	369	26·50	756	56·50	1600	12·25	347	24·00	682
Females .	20	31·00	879	7·50	214	18·50	526	22·00	624	10·50	298	15·50	488

or two cases. The weight of the lungs in these cases is given in Table II.

On comparing this Table with that showing the weight of the lungs in all the 100 cases dealt with in this paper, it is seen that the aggregate effect of whatever pathological changes occur in these organs during general paralysis, is to increase their average weight by about 2¼ ounces, or 122 grammes, in the male sex, and by about 1 ounce, or 27 grammes, in the female sex. A comparison, however, of the weight of the normal lungs in general paralytics, with the weight of the normal lungs in persons not affected by that malady, shows that during the course of uncomplicated general paralysis the lungs tend to diminish in weight. According to Dr. John Reid's observations, the average weight of the right lung is 24 ounces in the male, and 17 ounces in the female; and the average weight of the left lung is 21 ounces in the male, and 15 ounces in the female. But the average weight of the right lung in the cases included in the Table here submitted, is 22¼ ounces in the male, and 17½ ounces in the female; while the average weight of the left lung is 19½ ounces in the male, and 15 ounces in the female. In female general paralytics it would seem that the lungs are, as regards weight, fully up to the healthy standard (in the case of the right lung the weight given in this Table exceeds that given by Dr. John Reid by half-an-ounce), but it is to be noted that the average quoted is derived from the weight of the lungs in only two female general paralytics, who were both considerably above average height, and cannot, therefore, be regarded as trustworthy. In male general paralytics, however, the diminution in the weight of the lungs is very decided, amounting to an ounce and three-quarters, on an average, in the right lung, and an ounce and a half in the left. This loss of pulmonary substance in general paralysis, when no actual disease has invaded the lungs, must be regarded as in some degree analogous to the atrophy which is seen in advanced age. Senile changes are indeed anticipated and exaggerated in general paralysis, in which a general failure of nutrition is witnessed in muscles, bones, viscera, and indeed in every organ and tissue. An abnormal trophic innervation is doubtless responsible for much of the impaired nutrition that is seen in this disease, but in the case of the lungs it is scarcely necessary to resort to any trophic theory to account for some wasting in them. It is to be remembered, that in the latter stages of this disease locomotion and vocalization are greatly interfered with, and sometimes abolished, while there are at the same time failure of the heart's action and weak inspiratory power. In protracted and uncomplicated cases, it may be shown during life that the chest's capacity is diminished, that the movements of the thoracic walls are restricted, and that the respiratory murmurs are feeble; and it is not to be wondered at, therefore, that some diminution of volume and change of consistence are found in the lungs after death. These organs are of pale colour, of dry texture, and less firm and resisting than they ought to be, having lost their elastic resilience, so that they are capable of being squeezed into a small compass. The air vesicles are of large size and the bronchi look as if thinned. The process upon which these changes depend is, in all probability, essentially one of withering and obliteration of capillaries dependent on diminished respiratory function and blood volume.

The other pathological changes encountered in cases of general paralysis, may be best considered as they affected the pleural covering of the lungs or the lung substance, as congestion, consolidation, or phthisical degeneration.

Changes in the Pleuræ.—The pleural sac enclosing the lung is more given to morbid affections than any other serous membrane, and in persons labouring under general paralysis it seems to be even more prone to abnormal conditions than amongst mankind generally. Such abnormal conditions, which were almost invariably the result of inflammation, were found in 64 of the 100 cases of general paralysis which I have made the subject of special examination as regards the state of the lungs. The nature and distribution of these abnormal conditions are indicated in Table III.

From this Table it appears that in 36 general paralytics out of 100, the pleuræ were in a normal state and free from adhesions, but that in the remaining 74 cases pathological changes of one kind or another were noted. In 5 cases there were unmistakable signs of recent inflammation of one or both pleuræ, and in 71 cases there were changes in the pleural sacs which might be fairly regarded as products of past inflammatory attacks. In those cases in which the adhesions were few in number and limited in extent, it might be said that they were not necessarily the result of inflammation properly so-called, but of the hyperplastic and proliferative processes which are so common in serous membranes, and which leave behind them pleural adhesions in so large a proportion of persons who have passed middle life; but even in those cases included in this Table, along with the whitish bands or patches of adventitious membrane connecting the pleuræ, there were in many instances changes in the pulmonary substance beneath, which pointed to their origin in inflammatory lymph. And in the much greater number of cases, in which the adhesions were wide-spread, or general and firmly organized, the conclusion was irresistible that they had arisen out of pleurisies, which had often perhaps attracted little or no attention. Of course, in a large proportion of these cases, a proportion which, however, it was impossible to estimate, the pleurisy had occurred before the incursion of the general paralysis; but still the facts, that traces of 5 recent pleurisies were found in 100 subjects, and that many of the adhesions which are tabulated as old were only comparatively so, and had probably been formed within two

TABLE III.

Showing State of the Pleuræ in One Hundred Cases of General Paralysis.

State of Pleuræ.	Males.	Females.	Total.
Pleuræ on both sides, free from adhesions and normal	30	6	36
Old, slight, and scattered adhesions on both sides	13	5	18
Old, strong, and general adhesions on both sides	8	4	12
Old adhesions confined to right side	19	4	23
Old adhesions confined to left side	5	1	6
Recent adhesions on both sides	1	-	1
Recent adhesions on right side	1	-	1
Recent adhesions on left side	1	-	1
Fluid in the pleural cavities	2	-	2
Total	80	20	100

years of the time of death, justify the belief that there is a special proclivity in general paralysis to inflammation of the pleural membrane. As will be seen presently, the sufferers from general paralysis are particularly liable to attacks of pulmonary congestion, which may readily spread to the pleuræ. They are in the prime of life when pleural inflammations are most frequent, and they are apt heedlessly to expose themselves to changes of temperature and currents of cold air. Anstie thought that extreme muscular over-exertion and strain, as in public speaking, sometimes produces pleurisy in otherwise healthy persons, and if this supposition is correct, it is easy to understand why pleurisy should be common amongst general paralytics, who, when excited, in the earlier stages of their malady, will talk or shout incessantly for hours, and even for days and nights together, with an energy and persistency that the most inveterate orator never attained to. And perhaps another explanation of the frequency of pleurisy in general paralytics may yet be discovered in some constitutional tendency to which both of these diseases are etiologically related.

When the adhesions between the pleura were old they were composed of tough white connective tissues; when they were recent they were formed of thick layers of yellowish lymph; and where effusion had taken place, the fluid was turbid and contained purulent matter and flakes of lymph.

It will be observed that where the pleuritic adhesions were confined to one side, they were located on the right side more frequently than on the left, in the proportions of four to one.

Congestion of the Lungs.—Of all pathological changes in the lungs in general paralysis, congestion is the most common, and this is often present to a degree much greater than one is accustomed to find after slow death from exhausting bodily diseases. It was present to a marked extent in 49 out of the 100 cases of general paralysis which are here dealt with, and in many of these its intensity was such that it was difficult to say that it did not amount to pneumonic consolidation. In 38 of the 49 cases, it existed in both lungs, in 11 cases it was limited to the right lung and in 6 to the left, and whether involving one

TABLE IV.

SHOWING THE WEIGHT OF THE LUNGS IN THIRTY-TWO CASES OF GENERAL PARALYSIS IN WHICH THESE ORGANS WERE BOTH CONGESTED.

—		RIGHT LUNG.						LEFT LUNG.					
		Highest.		Lowest.		Mean.		Highest.		Lowest.		Mean.	
		Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.
Persons .	32	35·00	992	12·00	340	24·50	698	31·75	903	14·00	395	22·50	641
Males .	24	35·00	992	17·75	505	27·25	773	31·75	903	16·25	460	25·00	709
Females .	8	21·25	602	12·00	340	17·75	505	18·75	530	14·00	395	15·25	435

or both lungs it was invariably more extreme, and extensively diffused in the male than in the female sex. In Table IV. is set forth the weight of the lungs in those cases in which both were congested, and here we see that the vascular engorgement and œdema caused an increase of weight of 5 oz. in the right, and of 5½ oz. in the left lung in the male, but of only a quarter of an ounce in each lung in the female. This observation is interesting in connection with the well-known fact, that women die of pneumonia in much smaller proportion than men do, and that that disease is, as a rule, much milder in them than in the other sex.

The congestion of the lungs in general paralysis is of course hypostatic in character, and is almost invariably most marked posteriorly, and in the lower lobe. But it is by no means limited to this pulmonary region, for in 25 of the 49 cases in which it existed, it is described as having extended throughout the whole lung. In 22 cases it was confined to the lower lobe, and in 2 cases (in one of which it was associated with an abscess, and in the other with miliary tubercles) it involved the upper lobe alone.

The amount of congestion varied from slight hyperæmia, with some serous infiltration of the pulmonary parenchyma, up to extensive engorgement of a dark red, or even blackish colour. The boundary line separating the cases of most pronounced congestion from those of actual pneumonia was certainly not well defined, and some of the cases tabulated as congestion might have been perhaps not improperly called pneumonia. The presence of even obscure crepitation in the congested lung tissue, the escape of frothy fluid from it on section, and the restoration in some degree of the natural colour of the lung by prolonged washing, were taken as signs that congestion had not actually passed over into the pneumonic condition. With the congestion there was constantly bronchial catarrh. In 17 cases pus was found in the bronchi, and in 30 cases there was frothy mucus with increased vascularity of their lining membrane.

The hypostatic congestion of general paralysis is not difficult to account for. The sufferers from that protracted and debilitating disease have their muscular strength reduced to a low ebb, and in them respiration is carried on, as we have seen, with little vigour. Incapable in many instances of voluntarily changing their position, they remain for prolonged periods in attitudes favouring the accumulation of blood in dependent parts, while a loss of power in the nervous mechanism, regulating the calibre of the vessels, is also conducive to local congestions. It is possible that in the advanced stages of general paralysis, there may be some degeneration of the excito-motor ganglionic elements, which exist in the plexuses surrounding the pulmonary vessels, in the inferior thoracic or superior cervical ganglion, in the communicating nerves, in the antero-lateral columns of the cord or in the vaso-motor centre in the medulla oblongata. But it is not necessary to suppose changes in any of these in order to explain the vaso-motor disturbances, which are evident at an early period in the course of general paralysis, and which some have regarded as an essential element in the disease, for recent experiments have demonstrated, that the vaso-motor centre in the medulla is linked with certain portions of the cerebral cortex. Budge has proved that irritation of the crus-cerebri induces arterial contraction, and Eulenburg and Landois have shown that definite areas of the cerebral hemispheres in dogs, corresponding to the central convolutions in man, constitute vaso-motor centres for the extremities of the opposite half of the body. These vaso-motor centres, destruction of which by the cautery leads to a more or less enduring increase of temperature in the limbs of the opposite side, are situated in the vicinity of the voluntary motor centres of the cortex, which are invariably involved in the degeneration of general paralysis. And if vaso-motor centres for the limbs are located there, it may be legitimately inferred that vaso-motor centres for the head and neck, thorax and viscera are not far off, in the irritation or destruction of which ample explanation is to be found of the pallors, flushings and perspirations, the local changes of temperature and the internal congestions which are so frequently noticed in general paralysis.

A practical lesson may be derived from the great liability of those who labour under general paralysis, to suffer from pulmonary congestion, and it is to this effect, that their days may be prolonged by careful attention to their decubitus. A mere change of posture may remove an hypostatic congestion which would soon deepen to such an extent as to curtail life, were the position of the body left unchanged, and careful nursing, by night and day, may do much to ward off complications of this kind. General paralytics who are reduced to helplessness should be systematically moved from side to side, when in bed, and from one attitude to another when occupying a sofa or chair.

It seems to me also, that the liability of general paralytics to pulmonary hypostasis affords a strong argument against that re-introduction of mechanical restraint into our asylums, which finds some advocates in these days. Were mechanical restraint sanctioned, it would probably be had recourse to more frequently in the case of general paralytics than in that of any other class of lunatics. The reckless violence and destructiveness which so often occur at the outset of the disease, and the tottering restlessness of its closing scenes, with the risk of fractured ribs which attends its whole course, would all recommend the employment of what is, at any rate, a sure method of avoiding some distressing accidents. But it is just in general paralysis that special dangers would wait on this sure method. In the stage of excitement there is the danger of pulmonary fluxion, and in the stage of dementia that of pulmonary stagnation. Bonds restraining the muscles are very apt to induce pulmonary fluxion where there are nervous erethism and palpitation, and Niemeyer speaks of scandalous cases of frequent occurrence, in which delirious persons, or patients with delirium tremens, having been brutally strapped to their beds, are found dead the next morning with bloody foam upon their lips. Then, appliances preventing free movement may cause, it is scarcely necessary to point out, passive congestion and œdema of the lungs, while the trouble of removing and reapplying restraint would inevitably interfere with those frequent changes of posture which are so desirable in advanced cases of general paralysis.

Pneumonia.—Distinctly pneumonic conditions of the lungs were found in 13 out of the 100 cases of general paralysis, the post-mortem reports of which we have been examining.

Two of the 13 cases might be regarded as simple or ordinary acute pneumonia with profuse fibrinous exudation, and the remaining 11 as instances of broncho-pneumonia and hypostatic pneumonia. Both lungs were involved in the inflammation in 8 cases, the right lung alone in 4 cases, and the left lung alone in 1 case. Only in 1 instance was a pneumonia found in the body of a female; 12 of the patients who laboured under this disease at the time of death being males. The increase in the weight of the lungs caused by the pneumonic process was very remarkable. In one male, in whom both lungs were in a state of gray hepatization, the right one weighed 46¾ oz., or 1326 grammes, and the left 49¾ oz., or 1407 grammes. In another male, in whom the left lung was in a state of red hepatization, that organ weighed 56½ oz., or 1600 grammes, while the sound lung of the other side weighed 23¾ oz., or 670 grammes. The mean weights of the lungs in the 8 cases (all males), in which both lungs were inflamed, was 35½ oz., or 100 grammes, for the right, and 33 oz., or 936 grammes, for the left lung.

As has been pointed out, in these cases of pneumonia, both lungs were inflamed in a large majority of instances, and in this respect the pneumonia of general paralysis resembles the congestive pneumonia described by Mr. Erichsen as the commonest cause of a fatal issue after capital operations, when of course, debility, prolonged recumbency, and nervous shock, combine to favour its occurrence. In Mr. Erichsen's cases the right lung alone was affected three times, the left lung alone also three times, and both lungs together, twenty-two times. But in ordinary sthenic pneumonia the right lung is most frequently affected, the left lung next, and lastly, both lungs conjointly. Thus Huss found that in 53 per cent. of the 2,616 cases included in his statistics the pneumonia was on the right side, while it was on the left side in 32 per cent., and on both sides in 15 per cent. Grisolle states that in 1,430 cases of pneumonia, which he collected from various authors, the inflammation was in the right lung 742 times, in the left lung 426 times, and in both lungs 262 times. But while, contrary to the ordinary rule in the pneumonia of general paralysis, as well as in the pulmonary congestion of that disease, both lungs are involved much oftener than either lung singly, it is to be borne in mind that when one lung alone is affected by inflammation or congestion, it is the right lung that most frequently suffers. Rosenbach has shown[1] that in acute diseases of the respiratory organs in cases of hemiplegia, the lung of the paralysed side is much oftener the seat of disease than that of the sound side. In trying to account for this, he discovered that the direct reflex irritability of the involuntary muscular tissue of the paralysed side is decidedly lowered, one result of which is that foreign matter, such as saliva, entering the bronchi of the paralysed side does not give rise to sufficient expulsive efforts, so that unilateral broncho-pneumonia is set up. Founding on Rosenbach's observations, it might perhaps be argued that, as in general paralysis the left or leading hemisphere of the brain suffers probably earlier and more profoundly than does the right hemisphere, and as lesions of the cortex always cause functional and organic derangements on the opposite side of the body, so the right lung must inevitably suffer oftener than the left one. But this theory affords no clue to the preference of pneumonia for the right lung in persons in whom there are no lesions of the cortex, and some more general explanation is yet needed of the right side tendencies not only of pneumonia and congestion of the lungs, but also of pleurisy.

When seeking for an explanation of the great prevalence of pneumonia in general paralytics, the classical researches of Traube at once come to mind. Traube demonstrated that section of the vagi in the neck in animals, produces inflammation of the lungs and early death; and he referred the pneumonia which so frequently follows the operation to paralysis of the digestive canal from the throat to the stomach, and the consequent accumulation in the œsophagus, and overflow or aspiration into the air passages, of the secretions of the mouth and particles of food. Steiner subsequently showed that ligature of the œsophagus at its middle point, with section of the recurrent laryngeal nerves, produced exactly the same results which Traube obtained on section of the vagi; and then, dividing the vagi longitudinally into two bundles, which he was able to differentiate as motor and sensory, he further showed that section of the motor bundle was followed by an extensive pneumonia in a few days, due, like the pneumonia following complete section of the vagi, to paralysis of the œsophagus and larynx; and that, while section of the sensory bundles alone did not induce pneumonia, but only irregularity of respiration, their section immediately subsequent to section of the motor bundles played some part in the succeeding inflammation, by leading, through the powerful aspiratory efforts induced, to the carrying into the bronchi of more fluid than would otherwise have reached them. Now, in almost all general paralytics in the last stage of the disease, there is more or less paralysis of the œsophagus and larynx, with accumulation of food and fluid in the pharynx, and imperfect closure of the glottis. Choking is a common occurrence amongst them, and has to be specially provided for, and it might seem, therefore, that the mechanical or non-trophic theory of the causation of broncho-pneumonia was fully applicable in their case. The objection to this theory that particles of food are rarely or never found in the bronchi of the inflamed lungs is disposed of by the observations of Frey[1] that the mere trickling or injection of saliva into the smaller bronchi is sufficient to set up inflammation. But while fully admitting that mere mechanical irritation from an *error loci* of the ingesta or secretions may play an important part in the causation of pneumonia in general paralysis, it must, I think, be maintained that there are other factors in its etiology which must not be lost sight of. Solutions of nitrate of silver have been injected into the bronchi of persons free from nervous disease, without setting up pneumonia, and amongst general paralytics cases of broncho-pneumonia occasionally happen which can be distinctly traced to a reflex cause, such as exposure to a cold draught. It is probably with the pneumonia of general paralysis very much as it is with the bedsores in that disease, of which the etiology is complex. Mechanical irritation by frequent wetting and prolonged pressure, is highly instrumental in inducing these bedsores, and great care in avoiding mechanical irritation may effectually prevent their occurrence. In the Royal Naval

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[1] 'Centralblatt,' 1879, No. 16.

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[1] 'Lo Sperimentale,' Jan. 1878.

Hospital at Yarmouth, where a large number of severe cases of general paralysis are always under treatment, a bedsore is never seen, and the officers pride themselves on their skill in preventing their occurrence. But on the other hand, no one who has studied the bedsores which occur in such cases, will doubt that they are produced by a degree of mechanical irritation which would have no such effect in a healthy person, and that they are sometimes developed with a rapidity which betokens a profound disturbance of innervation in the part, as does also their tendency to dangerous extension. And so in the pneumonias, as in the acute and chronic decubitus of general paralysis we are forced to assume that besides mechanical irritation, some centric nervous influence is at work, causing neuro-paralytic hyperæmia, and perhaps a suspension of healthy trophic influences, for the existence of trophic fibres in the lungs is not yet finally disproved.

Phthisis.—Phthisical consolidation, or disintegration of lung tissue, was observed in 25 out of the 100 cases of general paralysis analysed, and in the subjoined Table, some information as to the weight of the lungs in these cases is supplied. The effect of phthisical disease is obviously very materially to augment the weight of the lungs which it invades. The mean weight of the lungs included in Table V. is not only much above the mean weight of the normal lungs in general paralytics, but considerably exceeds the mean weight of the lungs when congested. The mean weight of the right lung in the phthisical cases, was 6 oz., or 166 grammes, in the male, and 2½ oz., or 66 grammes in the female—greater than the mean weight of the right lung in the normal cases, and 1 oz., or 27 grammes, in the male, and 2¼ oz., or 60 grammes, in the female, greater than the mean weight of the lungs in the cases in which there was congestion. The mean weight of the left lung in the phthisical cases was 5¾ oz., or 167 grammes, in the male, greater than the normal standard, but in the female the mean weight of this lung was a trifle short of that standard. As compared with the cases in which there was congestion, the mean weight of the lungs in the phthisical cases was slightly in excess in the male, and slightly deficient in the female.

TABLE V.

Showing the Weight of the Lungs in Twenty-Five Persons Labouring under General Paralysis and Phthisical Disease.

—		Right Lung.						Left Lung.					
		Highest.		Lowest.		Mean.		Highest.		Lowest.		Mean.	
		Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.	Ounces.	Grammes.
Persons .	25	40·75	1155	7·50	214	25·50	725	32·00	905	10·50	298	22·00	621
Males .	17	40·75	1155	20·75	590	28·25	800	32·00	905	15·00	425	25·25	718
Females .	8	27·00	765	7·50	214	20·00	565	20·50	578	10·50	298	14·75	417

In six of the cases included in Table V., only the remnants of past phthisical disease were found in the lungs. These consisted of nodules of earthy or chalky matter, situated at the apex of the lung in every case, and occupying one apex in four, and both apices in two cases. Around these nodules or masses, which were single in four cases and double in one, and which varied in dimensions from the size of a pea to that of a walnut, the lung tissue was invariably dark and indurated, while over them the pulmonary surface was more or less puckered. As it is highly improbable that resolution with absorption of intra-alveolar exudation, and drying up of tuberculous deposits, could take place during the progress of so debilitating a malady as general paralysis, it may be fairly inferred that in these cases the phthisical affection had existed, and had been practically cured long before the supervention of the cerebral disease. In the remaining cases, however, in which phthisical changes were found in the lungs, these were of a kind indicating their recent origin and active state. They were confined to the right lung in 3 cases (all males), to the left lung in 7 cases (4 males, and 3 females), and involved both lungs in 9 cases (5 males and 4 females). They consisted in grey miliary tubercles in 9 cases, in caseous masses in 5 cases, and in mixed grey granulations, and yellow cheesy masses in 5 cases. Cavities were associated with the miliary tubercles in 3 cases, and with the mixed tubercular and caseous degeneration in 4 cases, being in 5 cases confined to the upper lobe, and in 1 to the lower lobe. The vomicæ were always of comparatively small size, with ragged walls, presenting no appearances of a false membrane, and the lung tissue around the miliary tubercles and nodules was for the most part congested and œdematous, and but little indurated. There was no room for doubt that in all these 19 cases, the inflammatory processes leading to phthisical consolidation, and destruction of lung tissue, had arisen during the course of the general paralysis, and had been cut short by the natural termination of that disease. In none of them had the disorganisation spread to that extent which we are accustomed to see in the lungs of patients who have died of phthisis.

Now the age at which patients who perish by general paralysis are attacked by it, and their previous history and physical characteristics, alike forbid us to attribute any phthisical disease which may be developed in them to the influence of hereditary predisposition. They have passed the time of life at which hereditary phthisis most frequently displays itself, and they have never exhibited that constitutional feebleness which is so characteristic of those who have an inherited tendency to that disease. They are as a rule vigorous and much enduring, until general paralysis lays its grasp upon them; we must therefore look for the personal factor in the causation of phthisis, when they suffer from it, not in an inherited, but in an acquired state of health, unless, indeed, certain neurotic tendencies which the sufferers from general paralysis and phthisis both exhibit, be regarded as having an etiological relation to the latter disease. And this acquired state of health is readily recognised in the general debility and impairment of power which constantly accompany general paralysis. Here we have that condition of the system which favours the development and progress of phthisis, when inflammatory changes are set up by any irritation or injury of the pulmonary tissue. And such irritation or injury is of course peculiarly likely to occur in the victims of general paralysis, whose mental disquietude and muscular helplessness, are alike apt to lead to chilling of the body by exposure to cold, and who are, as we have seen, exceedingly susceptible to pulmonary congestion and bronchial catarrh.

But there is another respect in which general paralytics may be regarded as favourably situated for contracting phthisis. They are generally, during their latter days at any rate, in close association with persons suffering from this disease, and they inhabit buildings in which it has been long established. Dr. Villemin, when speaking of the extraordinary prevalence of phthisis amongst soldiers, expressed his conviction that the barrack is to the soldier, in the production of consumption, what the regimental stable is to the horse in the development of farcy; and evidence is now accumulating daily in support of the transmissibility of phthisis from one person to another.

The theory of Koch, that the tubercle bacillus is essential to the development of the phthisical process, and that phthisis is therefore a communicable disease, although not fully established, is daily becoming more probable, and the history of the connection of phthisis with general paralysis seems to lend it some confirmation. The prevalence of phthisical destruction of lung tissue amongst general paralytics is very remarkable. In my cases it existed in 19 per cent. But the prevalence of phthisis, not merely amongst general paralytics, but amongst the insane generally, is noteworthy. While in the population of England, above twenty years of age, phthisis is responsible for 14 per cent. of the whole mortality, in the public lunatic asylums of England and Wales, the population of which may be taken as above twenty years of age, phthisis is the assigned cause of death in 15 per cent. of the whole mortality; while it exists at the time of death in a considerable proportion of cases of general paralysis, epilepsy, &c., in which it is not the certified cause of death, and so does not appear in the tables of mortality.

Now a certain proportion of patients, when admitted into lunatic asylums, are labouring under phthisis, but, on the other hand, a certain proportion of patients are discharged from these establishments when labouring under that disease; and it is, I believe, incontestable that phthisis is generated in lunatic hospitals to a greater extent than in the general community. Bearing in mind that the inmates of asylums are well fed, clothed, and housed, and live in situations specially selected for their salubriousness, and under strict hygienic regulations, the prevalence of phthisis amongst them strongly suggests the propagation of the disease by contagion. And this suggestion has additional probability given to it by several other facts and considerations. While in the general community the mortality of males from phthisis is greater than that of females, in lunatic asylums the reverse is the case. In England and Wales, phthisis is the assigned cause of death in 15·2 per cent. of the whole mortality amongst males, and in 13·4 per cent. of the whole mortality amongst females; but in the pauper lunatic asylums of England and Wales it is the assigned cause of death in 12 per cent. of the whole mortality amongst males, and in 17 per cent. of the whole mortality amongst females. Now, the chief respects in which female lunatics differ from male lunatics in their mode of life, are that they are more sedentary in their habits, and are less out of doors, being more confined to their day-rooms and wards, and compelled to breathe more constantly the air which is breathed, and possibly poisoned, by the patients who are consumptive. And further than this, it might be shown that the mortality from phthisis has been materially diminished in some asylums by increased attention to sanitation, the ventilation of the buildings, and the out-door exercise of the patients, by the adoption, in fact, of means calculated to lessen the risk of dissemination of contagious or infective matter in the atmosphere. It may also be shown, that in every asylum phthisis originates most frequently in patients inhabiting wards such as the infirmary wards, in which the air is liable to be contaminated by the breath and sputum of phthisical patients. And thus it is, I should be inclined to maintain, that phthisical conditions are so common in general paralytics. They are, for the most part, from the first, unfit for out-door employment, and in the latter stage of their illness they are, almost without exception, relegated to the infirmary wards, so that they may have the benefit of better nursing. But in these wards they are very generally in association with patients sinking under phthisis; and the fact that 19 per cent. of general paralytics dying in asylums present evidences of recent phthisical disease, whereas phthisis is responsible for only 15 per cent. of lunatic asylum mortality as a whole, seems to me to support the view that phthisis is communicable, and is dependent on organisms which are specific. That the tubercle bacillus is a constant attendant on tubercular processes cannot now be denied, that it is the true cause of their infective property is most likely, and that it may be communicated from man to man, and set up phthisical changes anew whenever it lights on a congenial soil, is rendered probable by facts such as those which have been adduced regarding the connection of phthisis with general paralysis.

Until Koch's theory is disproved, it would be prudent to act on the assumption that it is true, and to prevent the close association of persons actually suffering from phthisis with those who, from inherited tendency or deterioration of health, are especially liable to contract the disease. A large number of lunatic asylums have now detached hospitals for contagious diseases, which fortunately stand empty for a great part of the year, and it might be well to isolate in those buildings all cases of phthisis. The experiment could do no possible harm, and there is every prospect that it would be attended with benefit to the victims of phthisis, and with safety to those who are in danger of its attacks.

Abscess of the Lung.—In 2 cases out of the 100, abscesses of the lung were discovered. In one of these the abscess, which was of the size of a small orange, and situated in the upper lobe of the right lung, was clearly the consequence of a pneumonia, but in the other, the abscesses (for there were three of small size) were probably of metastatic origin. They were in the lower lobe of the right lung of a man, in whom extensive bedsores had formed almost suddenly on the sacrogluteal region a few weeks previously, and who had suffered from remittent fever. There was no tubercular matter in either lung in this man, and each abscess had surrounding it a ring of consolidated pulmonary tissue.

Gangrene of the Lung.—Dr. Mickle has seen gangrene of the lung in general paralysis, but nothing approaching it was observed in any of the 100 cases which I have been analysing.

SPONTANEOUS POSTURES OF THE HAND CONSIDERED AS INDICATIONS OF THE CONDITION OF THE BRAIN.[1]

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The term posture is here used as it is commonly understood, to indicate the relative position of the several members of the body with regard to one another, or the relative position of the individual parts of a member.

It is proposed to speak in this paper of only a few spontaneous postures of the hand.

The postures of a limb depend in their immediate mechanism upon the resultant action of opposing muscles, the relative tone of the antagonistic flexors and extensors, the adductors and abductors, etc. Various views may be held with regard to the nerve-mechanism which regulates the contraction of opposing sets of muscles. That mechanism may be considered a line of reflex action regulating the action of the muscles, or the balance may be considered due to some other kind of nerve-mechanism. In any case it will, I think, be granted that some portion of the central nerve-system is the cause of that balance of the muscles that produces the posture, and therefore the posture is an index of the condition of that central nerve-mechanism.

In examining the condition of the nervous system in children, it has for several years been my habit to observe what spontaneous posture would be assumed by the hand when the forearm was held out.[1] Let a weak, nervous child be requested to hold out her hands in front on a level with the shoulder in the prone position. The limb is now free or disengaged, and the posture assumed by the hand is in most cases, I believe, the spontaneous outcome of the action of the nerve-centres.

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[1] Read before the Royal Medical and Chirurgical Society of London Nov. 28th, 1882.

Let me describe the spontaneous posture of the hand usually seen in a nervous child, and then discuss the causation and meaning of this posture.

In this posture—"the nervous hand"—the wrist is slightly flexed, or bent, the metacarpo-phalangeal joints are moderately hyper-extended (extended beyond the straight line), the first and second internodes being either slightly flexed or kept straight. The thumb is extended backwards, and somewhat abducted from the fingers. This spontaneous posture I have seen, and others with me, in hundreds of cases, usually in nervous children, bad sleepers, those convalescent from chorea, etc.; the same spontaneous posture is sometimes seen in partial hemiplegia. The posture is often bilateral, but is usually unequally represented on the two sides, it is also often seen on one side only, especially in children convalescent from hemichorea. From continued observation of spontaneous postures, and after frequently trying what spontaneous posture would be assumed by the free hand in various subjects, cases of weakness, strong men, feeble women, nervous children, an empirical idea was obtained as to the indication of each posture. Then comparison, analysis and analogy enabled me to suggest some definite principles. In seeking a rational explanation of this posture, I first tried to find a solution by looking at it after the manner in which Charles Darwin made most of his observations upon postures. Cases were looked for where this posture of the hand was assumed or brought about for some useful purpose, or its occurrence was attended with some kind of amelioration of weakness. Then this "nervous hand" was seen (in a hand not free or disengaged) in people who, standing, lean a little forward with the outspread hands resting on the table for support. This hand seeking rest droops at the wrist, and then if the stimulus to the muscles is weak, as the weight of the trunk bears upon the resting hands, hyper-extension of the metacarpo-phalangeal joints, and of the thumb, is mechanically brought about. If this be accepted as an explanation, the argument stands thus:—

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[1] See 'BRAIN,' Part XI.; and 'British Medical Journal,' Dec. 6, 1879.

1. Observation shows that when a man is tired or weak he often rests his hands as described.

2. The nerve-mechanism corresponding to the hand is thrown into the special condition corresponding to the "nervous hand," and thus that posture is mechanically brought about.

3. The nerve-mechanism corresponding to the hand falls spontaneously into the condition corresponding to the "nervous hand" when the man is tired, or weak.

4. When the hand of a weak man is held out free, its nerve-mechanism spontaneously places it in the "nervous posture."

The attempt at explanation may be made again, commencing with the principle of analysis and then applying the principle of analogy.

Anatomical analysis shows that this "nervous hand" presents two principal elementary conditions:

(1) Flexion or drooping of the wrist.

(2) Hyper-extension of the metacarpo-phalangeal (small) joints.

So much for analysis, now as to analogy.

Looking for analogy between postures, flexion, or drooping of the wrist, may be seen as in a hand that is resting, in a hand that is passively held out while the patient is deeply asleep, or in deep coma, or in a paralysed arm. It is probable then that the wrist flexion indicates the weakness of the "nervous hand."

Hyper-extension of the metacarpo-phalangeal joints and thumb may be seen as a temporary condition in subsultus tendinum, and chorea. In all these cases the unstable condition of the nerve-mechanism is indicated by the extensor movements of small parts.

Now looking at the elementary conditions shown by analysis to constitute the "nervous hand," and the probable indication of the conditions analogous to these, we get some kind of explanation of this posture. The drooping wrist is analogous to that seen in hemiplegia; the hyper-extension of the knuckles and thumb is analogous to conditions such as chorea, and subsultus tendinum. This appears a more rational explanation than that first given, and the methods of analysis and analogy give some indication of the physiological significance of the posture. Something very like this posture of the hand is indicated by Dr. Little in his work on deformities, 1853, as the result of "spastic contraction."

Another posture of the hand less frequently seen in pathological states, I described under the name of the "Energetic hand," and since so doing I have found the posture figured by H. Meillet of Paris, with the note "Main dite du prédicateur emphatique. La Salpêtrière, service de M. Charcot, salle Saint-Paul, No. 6, Isméric Angot." It is there figured as a permanent deformity resulting from brain disease. Dr. Little also figures this posture as due to spastic contraction. In this posture the wrist is extended, and the small joints are all in flexion. Here analysis shows the large joint, the wrist, in extension, the opposite to the condition of weakness, and this extension of the large joint gives the posture the indication of excitement in the nerve-mechanism. The small joints are all flexed, as seen in the hand of a man in sleep or resting, and this gives the posture the indication of strength or activity with rest.

Anatomical analysis shows the "energetic hand" to be the antithesis of the "nervous-hand."

	Nervous Hand.	Energetic Hand.
Wrist	flexed	extended
Metacarpo-phalangeals	extended	flexed
1st and 2nd internodes	flexed	flexed
Thumb; metacarpo-phalangeal	extended	flexed
Thumb; internode	extended	flexed
Phalanges; relative position	slightly abducted.	adducted

The physiological expressions of the two postures are also opposites.

This suggests another principle of use in explaining the meaning of certain postures. The principle of antithesis[1] may be enunciated as follows:—"In opposite conditions of the nervous mechanisms producing the postures of a joint, the postures will be opposite or antithetical"; e.g., in a weak or inactive condition of the nerve-mechanism governing the wrist flexion results; this would lead us to anticipate that in an excited condition of this piece of nerve-mechanism we should find the wrist extended.

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[1] Charles Darwin uses the principle of Antithesis to explain many modes of Expression. See 'Expression of the Emotions,' p. 28.

My present object is to show that spontaneous postures may be studied with advantage as indications of the conditions of the brain. As to the principles involved in these inquiries. A primary postulate, involved in the following principles, is that—

"If we see some spontaneous nerve-muscular action often repeated in the same and in different subjects, it may be assumed that there is some nerve-centre, or nerve-mechanism, corresponding which can act independently." In any particular instance the assumption of the existence of such a centre or piece of nerve-mechanism would be strengthened if it could be shown that there are cases of excitation and cases of paralysis of such hypothetical centre.

In analysing any posture of the hand we may consider the bearing of the following Principles, or points of analysis, upon the subject:—

I. Describe the posture indicating the anatomical condition of each joint.

II. Consider the posture of the small parts of the limb as distinguished from the larger parts.

In many other fields of observation the importance of this rule has been illustrated thus:—the small parts of a member are the most affected in chorea, in hemiplegia, in subsultus tendinum.

III. Consider the different relative postures seen in the large and small joints.

Interdifferentiation of the postures of the large and small joints may give important indications: the large joint may be paretic while the small joints are in extension, from irritability of the nerve-centres which govern their movements. In such a case there would be an interdifferentiation of the nerve-centres governing the different joints.

IV. Consider the relative condition of posture in collateral joints. The knuckles are collateral joints, the posture of each may be different; so there may be differences in the posture of the collateral first inter-phalangeal joints of the fingers. Collateral interdifferentiation is usually seen in the movements of the hand in chorea, but sometimes all the fingers open and close together. Collateral differentiation is marked in athetosis.

V. Consider if the posture is symmetrical.

A posture may be called symmetrical when the parts of the member are equally placed or balanced about the axis of the limb. Symmetry therefore indicates the exact balance of opposing muscles, and consequently the exact balance of the portions of the nerve-mechanism which cause the postures of the limbs.

VI. Consider if there be any indication of excitement or over-action of any nerve-centre or centres usually weak, such as the centre or nerve-mechanism governing the extensor movement of a joint.

It is certain that extension of a joint is usually a movement of less power or strength than flexion of the joint. Observations, comparisons and analysis seem to indicate the nerve-centre, or mechanism governing the extension of a joint (i.e. the action of the weaker muscles) as being weaker than the centre governing flexion. Observe if such indications of excitement subside in sleep, they may do so in chorea; some cases lose the "nervous hand" during sleep, the hand when held out free falling into the posture of the "hand in rest."

VII. If there be general, equal, widespread stimulation or weakness of portions of the nerve-mechanism governing the postures of a limb, or part of the body, the action of the stronger muscles prevails. General weakness may be indicated by general preponderance of weak centres as in general extension. General weakness may also be indicated by general slight flexion, showing all the nerve-mechanism paretic and the stronger flexor muscles prevailing.

VIII. Consider in the posture if any joint or set of joints be in position analogous to that of any other posture, and whether the significance of the position of that joint be the same as in the posture to which the analogy is made.

IX. Consider if the posture be the antithesis of any other known spontaneous posture.

It is probable that in many cases opposite conditions of the nerve-mechanism producing postures will produce antithetical postures.

The following suggestions are offered as to the *methods of determining whether a posture is the outcome of the spontaneous action of the nerve-centres.*

A posture observed can only be considered as the outcome of the spontaneous action of the nerve-mechanism when the limb or part is free and disengaged. If the muscles of the hand are engaged in holding an object, or in an act of manipulation, the postures of the hand are not simply the outcome of the spontaneous action of the nerve-mechanism.

Postures are frequently seen accompanying, and apparently caused by, demonstrable brain-disease; as examples the "convulsive hand," cerebral facial palsy. When these postures occur otherwise, apparently spontaneously, they may be looked upon as nerve-muscular signs produced by the nerve-mechanism corresponding.

Postures which are frequently seen in very young children and infants are certainly spontaneous. Postures seen to be frequently repeated in many such young subjects are probably spontaneous nerve-muscular actions. With regard to postures thus observed in many infants it is probable that the piece of nerve-mechanism corresponding is well developed, and rendered very definite so as to be easily excited to spontaneous action.

When a posture seen in an adult is found to be analogous to one often seen in infants, the probability of its spontaneous origin is strengthened.

Postures of disengaged parts, still observed in man, and frequently represented in ancient art, are probably of frequent spontaneous occurrence, and highly characteristic nerve-muscular postures.

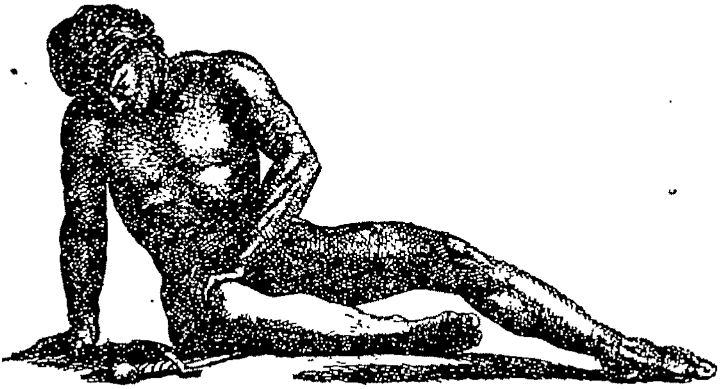
The application of the "principles of analysis" may assist in determining whether a certain posture observed is probably the outcome of the spontaneous action of the nerve-centres.

In these studies care must be taken to avoid certain errors from attributing all postures to the action of the central nerve-mechanism.

Postures may be due to joint-disease, especially Arthritis deformans. M. Charcot carefully differentiates between this deformity, and the posture he has described as the "writing hand" often seen in Paralysis agitans.

Postures may be due to local causes—tumours, local inflammation, etc.

Postures may be determined by organic conditions such as difficulties in the respiratory and circulatory organs causing orthopnœa. Sir. C. Bell drew attention to this matter in his critical analysis of the posture of the Dying Gladiator. Such postures are not the direct effect of the spontaneous action of the nerve-centres.



THE DYING GLADIATOR.
(After a drawing by Sir C. Bell.)

Gravity may be a factor in the causation of postures, whether the member be "free" or "engaged," thus: In the figure of Hercules at rest, he leans on a vertical club to support his body, and the posture of the right arm is determined mainly by gravity. In a paretic arm such as is often seen in chorea, or from brain disease, the wrist will droop into flexion if the forearm is held out prone, but will fall into extension if the forearm is held out supine. Dorsal decubitus results from gravity, together with general palsy of the motor nerve-mechanism.

Postures may be due to local nerve injury or disease, thus: Injury to the facial nerve; injury to the musculo-spiral nerve; a gumma pressing upon the third cranial nerve, may produce certain postures of the parts supplied by these nerves, such postures not depending upon the condition of the nerve-centres.

Postures may be due to rigid muscular contraction dependent upon permanent brain lesion, such as descending sclerosis. Such cases should be kept separate from the postures due to a temporary, it may be momentary, condition of the central nerve-mechanism. It is hoped that some proofs have been given that the study of spontaneous postures as indications of the condition of the brain is useful, and that it may be considered as one of the exact methods of studying the nervous system in its physiological and pathological conditions.

The subjoined tables contain descriptions of eight typical postures applying the "Principles" to the study of each.

POSTSCRIPT.

It seems desirable to give some further indication of the knowledge that it is hoped to gain from the systematic study of postures, and to give further descriptive details of the typical postures included in the tables. I do not attempt to indicate what special portions of the brain are concerned in producing different postures, and do not think such details can at present be given. I cannot localise a motor centre for the "hand in fright;" could we do so, it would afford some evidence as to what portion of brain mechanism is concerned in the mental condition called fright. In the case of an idiot, whose hands were usually in the convulsive posture, I took cast of the hands, and subsequently had the opportunity of examining the brain, which showed marked defects in the posterior convolutions. It is probable that difference in hand posture on the two sides indicates a different action of the two hemispheres, and of this I hope to give further evidence on another occasion founded upon the association of postures. When the hands are held free, a difference in the posture on the two sides, or different movements in the two hands, give

TABLE I.

Principles.	Nervous Hand.	Energetic Hand.	Hand in Rest.	Ortho-extended Hand.
1. "Anatomical analysis."	Wrist . . flexed. Metacarpo-phalangeals hyperextended. 1st and 2nd internodes flexed. Thumb, metacarpo-phalangeal . hyperextended. Thumb, internode hyperextended. Phalanges, relative position abduction.	Wrist . . . extended. Metacarpo-phalangeals flexed. 1st and 2nd internodes flexed. Thumb, metacarpo-phalangeal . . flexed. Thumb, internode flexed. Phalanges, relative position adducted.	Wrist . . . flexed. Metacarpo-phalangeals flexed. 1st and 2nd internodes flexed. Thumb, metacarpo-phalangeal . . . flexed. Thumb, internode flexed. Phalanges, relative position adduction.	Wrist . . ortho-extended. Metacarpo-phalangeals ortho-extended. 1st and 2nd internodes ortho-extended. Thumb, metacarpo-phalangeals . ortho-extended. Thumb, internode ortho-extended. Phalanges, relative position adducted.
2. "Small parts contrasted with large parts."	Small parts characterised by hyperextension of the metacarpo - phalangeal joints in contrast with flexion of the large joint —the wrist.	Small parts in flexion in contrast with the flexion of the large joint—the wrist.	Small parts in flexion. Large parts in flexion.	Small parts ortho-extended. Large parts ortho-extended.
3. "Interdifferentiation."	Interdifferentiation marked, the small metacarpals and the thumb being extended in contrast with flexion of the large joint —the wrist.	Interdifferentiation marked, the small joints being all flexed in fingers and thumb in contrast with extension of the large joint—the wrist.	No interdifferentiation in the type described.	No interdifferentiation in the type described.

TABLE I. (*continued*).

Principles.	Nervous Hand.	Energetic Hand.	Hand in Rest.	Ortho-extended Hand.
4. "Collateral differentiation."	No collateral differentiation in the type described.	No collateral differentiation.	No collateral differentiation.	No collateral differentiation.
5. "Symmetry."	The type symmetrical; the two hands may differ.	Symmetrical.	Symmetrical.	Symmetry of this type complete.
6. "Excitation of weak centres."	Metacarpo-phalangeals extended, indicating excitement of weak centres.	Wrist extended, indicating excitement of the weaker centre of a large joint.	No extension of any joint, indicating no excitement of weak extensor centres.	Perfect balance, no excitation or weakness. This is the normal strong hand.
7. "General excitement or weakness."	Cannot apply here, as there is interdifferentiation.	Cannot apply here, as there is interdifferentiation.	General flexion indicating predominance of the strong flexor muscles from general weakness of the nerve-centres.	Perfect balance, no excitation, no weakness.
8. "Analogy."	Wrist flexed as in "hand in rest." Knuckles and thumb in hyperextension as seen in chorea, in subsultus tendinum in some irregular twitches in sleep.	Wrist extended as in "hand in fright." Knuckles and fingers and thumb flexed as in "hand in rest."	The type of complete relaxation of all the nerve-centres.	The type of complete balance of all the nerve-centres, hence the type of strength without excitement.
9. "Antithesis."	The energetic hand.	The "nervous hand."	α. The "ortho-extended hand." β. The "hand in fright."	α. The "feeble hand." β. The "hand in rest."

TABLE II.

Principles.	Convulsive Hand.	Hand in Fright.	Feeble Hand.	Ortho-extended Hand with Thumb Drooped.
1. "Anatomical analysis."	Wrist flexed or extended, &c. Metacarpo-phalangeals flexed. 1st and 2nd internodes flexed. Thumb, metacarpo-phalangeal. . flexed strongly. Thumb, internode flexed strongly. Phalanges adducted.	Wrist . . hyperextended. Metacarpo-phalangeals ortho-extended. 1st and 2nd internodes ortho-extended. Thumb, metacarpo-phalangeal . ortho-extended. Thumb, internode ortho-extended. Phalanges abducted.	Wrist . . . . flexed. Metacarpo-phalangeals flexed. 1st and 2nd internodes flexed. Thumb, metacarpo-phalangeal . . . . flexed. Thumb, internode . flexed. Phalanges adducted, metacarpus contracted.	Wrist . . ortho-extended. Metacarpo-phalangeals ortho-extended. 1st and 2nd internodes ortho-extended. Thumb, metacarpal bone adducted or drooped. Thumb, internode ortho-extended. Metacarpus straight, phalanges adducted.
2. "Small parts contrasted with large parts."	Condition of the wrist varies (it may be flexed, extended, adducted).	Small parts ortho-extended, while the large wrist joint is hyperextended.	Small parts in flexion. Large parts in flexion.	.. ..
3. "Interdifferentiation."	Interdifferentiation various; the wrist posture varies.	No interdifferentiation, all joints extended.	No interdifferentiation; all joints flexed.	.. ..
4. "Collateral differentiation."	Probably no collateral differentiation in most cases.	No collateral differentiation.	No collateral differentiation in the type described.	Collateral differentiation of the metacarpal bones, the thumb being drooped.
5. "Symmetry."	.. ..	.. ..	Symmetrical.	Not symmetrical; the metacarpus being straight but the thumb drooped.

TABLE II. (*continued*).

Principles.	Convulsive Hand.	Hand in Fright.	Feeble Hand.	Ortho-extended Hand with Thumb Drooped.
6. "Excitation of weak centres."	All the hand in strong flexion, but the wrist may be extended, indicating excitement of this weak centre.	All the extensor weak centres are excited.	No excitement of any weak centres.	No excitation of weak extensor centres, but marked weakness of the extensors of the thumb.
7. "General excitement or weakness."	General excitement of all the nerve-centres causes the flexors strongly to predominate.	General excitement of all the extensor centres indicating general excitement of the weak centres.	General weakness.	Perfect balance of nerve-mechanism for fingers and wrist, weakness of the centres for the thumb.
8. "Analogies."	. .	α. Analogous to the "energetic hand," with excitement of the weak extensor centres of the digits. β. Analogous to the "ortho-extended hand," with excitement of the weak extensor centre of the wrist.	.. ..	.. ..
9. "Antithesis."	Metacarpus contracted as in the "feeble hand."	Hand in rest.	Metacarpus contracted as in the "convulsive hand." Ortho-extended hand.	.. ..

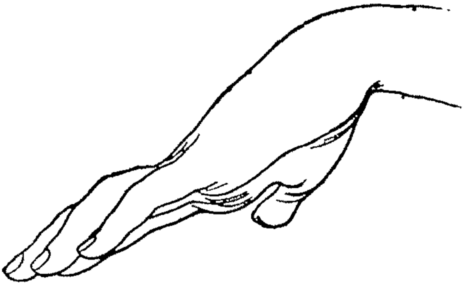


Fig. 1.—The Nervous Hand.

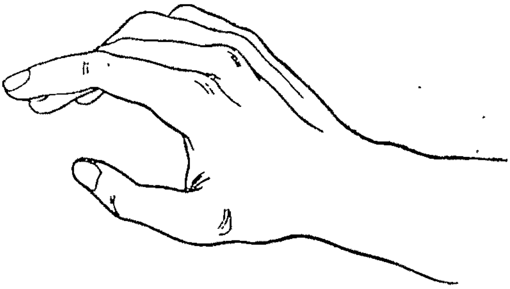


Fig. 2.—The Energetic Hand.

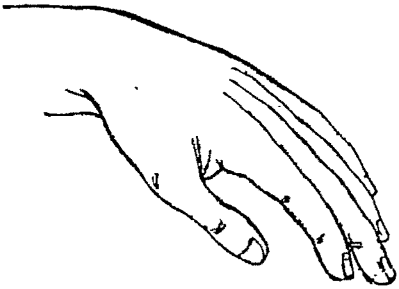


Fig. 3.—The Hand in Rest.

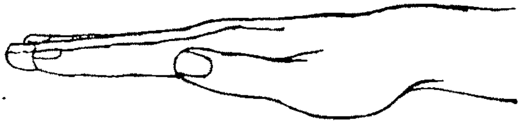


Fig. 4.—The Straight Hand.

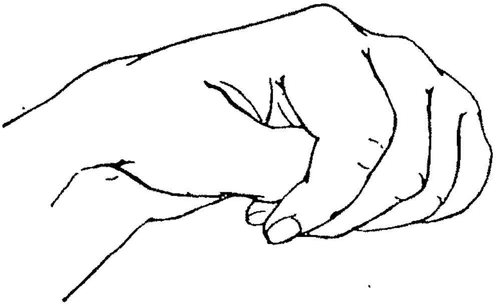


FIG. 5.—THE CONVULSIVE HAND.

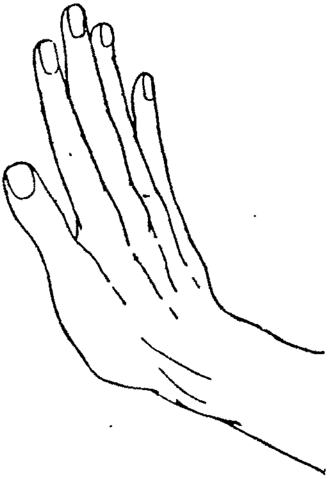


FIG. 6.—THE HAND IN FRIGHT.

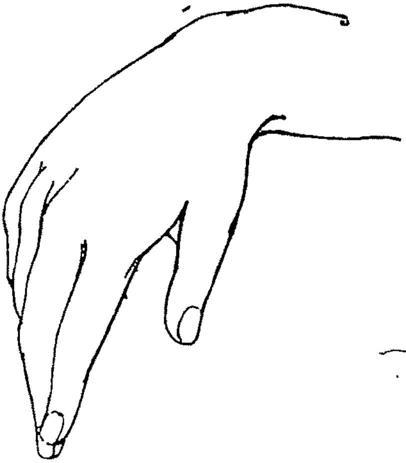


FIG. 7.—THE FEEBLE HAND.

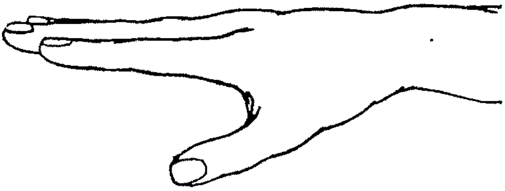


FIG. 8.—THE STRAIGHT HAND WITH THUMB DROOPED.

evidence that the two hemispheres are in a different condition. These studies show that in many people the nerve-centres are not strictly symmetrical in action, the average postures of the face and hands being asymmetrical, e.g., frequent one-sided, grinning, the nervous hand is often seen on one side only, usually the left; these observations may be conducted in healthy people, and give evidence of the individual's idiosynchronism. The study of postures gives examples of the application of the "Principles," enunciated above, and I think, affords some proof of their practical importance. Most of these points are commonplace clinical considerations, but principles vi. and vii. perhaps require further explanation and defence.

It may be readily admitted that it is desirable and important to know what are the motor indications of excitement or over-action of a nerve-centre, also to know the relative strength of the different motor centres, and what are the outward physical signs of wide-spread stimulation or weakness among portions of the nerve-mechanism. In numerous cases, where common experience has shown general strength, weakness, or wide-spread stimulation, etc., these principles of analysis have been applied, and some general knowledge has thus been obtained, but more exact knowledge is wanted. I trust by an experimental method, described in the 'Journal of Physiology,'[1] to be able to give hereafter a much more precise and definite account of these principles.

These studies afford evidence that some parts of the brain may be in a condition of weakness, while others are in a state of strength or excitement, and that one hemisphere may be weaker in action than the other. Again a change of posture may indicate a change in the corresponding nerve-mechanism, thus in chorea when the forearm is passively supported, we often see that the free hand passes from the posture of rest to the convulsive posture, thus indicating the change in the condition of the nerve-mechanism, corresponding to the muscles of the hand. By daily recording the most common involuntary postures of the free hand, we obtain indications of the daily condition of the nerve-mechanism corresponding.

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[1] Vol. iv. No. 2, August 1883.

A few words as to the hand postures analysed in the tables.

The ortho-extended hand.—A strong man when asked to put out his hand, holds it out straight, all parts in the same plane, no flexion or extension, but perfect balance. This statement is founded on numerous observations. I have often requested a body of students, or other healthy subjects, to hold out their hands, so also healthy children, who could know nothing of my object, and thus I have had the opportunity of judging that this well-balanced posture indicates the normal action of the nerve-mechanism employed in the act.

The hand in rest may be seen in a strong and healthy subject during sleep, also when the hand hangs free and quiet, as when doing nothing, and in a state of mental quiescence. I have often seen it during quiet conversation, and in men while walking or travelling. The metacarpus is often slightly contracted, but not to such a marked degree as in the "feeble hand."

The convulsive hand.—This posture is well known. Trousseau described it in his 'Clinical Lectures' as commonly caused by the involuntary contractions in tetany. The condition of the metacarpus is specially noteworthy. I have taken casts of several cases and have measured many more. It is as Trousseau described it, arched or contracted by approximation of the metacarpal bones, which are screwed together as in the hand of the accoucheur. Important variations of this posture might be described; it is often seen in various pathological states.

The feeble hand.—If the disengaged hand of a feeble child or woman is held out we commonly see a slight, but important, departure from the hand in rest. The thumb is drooped, and its metacarpal bone is approximated to the palm, all the metacarpals being bent round as in the convulsive hand, this is a posture often seen.

Independent of all direct benefit that may follow from observing movements and postures, which are the result of the last movements, I maintain that all physical phenomena which are the direct outcome of brain-action are worthy of observation, record and analysis.

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MAY TABES DORSALIS SOMETIMES HAVE A PERIPHERAL ORIGIN?

BY HERBERT W. PAGE, M.A. CANTAB., F.R.C.S. ENG.

The origin of the system diseases of the spinal cord is still profoundly obscure, notwithstanding that their clinical history and pathological changes have been carefully studied and recorded. Amongst them tabes dorsalis is one of the commonest, but its cause and origin are nevertheless wholly unknown. A history of syphilis is indeed often to be obtained from the victims of this disease, yet syphilis is far from being generally accepted as an undoubted cause, and were Duchenne de Boulogne now alive, it is improbable that he could materially alter the words which he wrote in his classical description of the malady many years ago: "Malgré le soin extrême que j'ai mis à étudier les antécédents des malades, je n'ai pu, dans quelques cas, entrevoir la moindre cause prédisposante ou déterminante." It is therefore not inappropriate perhaps to put the question which heads this paper, and even though the answer to it may be deemed inconclusive here, it may perhaps suggest a line of inquiry to other and more competent observers whose opportunities are greater than my own. I am still further emboldened to take the MS. out of the obscurity of a drawer, by the recent important contribution of MM. Pitres and Vaillard on non-traumatic peripheral neuritis, an account of which will appear in the "Abstracts of Foreign Journals."

Before attempting to answer the question, however, let me relate, and take as a text, the following case, that of a man who many years ago had "perforating ulcers" of the feet, and who now is the subject of tabes dorsalis, with a characteristic arthropathy of one joint.

F. G., aged 36, a spare, not unhealthy-looking man, presented himself as an out-patient at St. Mary's Hospital on Jan. 5, 1882, complaining that for the last two months the top of his left foot had been much inflamed and somewhat painful in walking. The dorsum of the foot was found red and œdematous, and he said that he had been occasionally troubled with the same condition ever since his great toe had been removed seven years ago. He further stated that his toe had been lost from frost-bite in India, where he was then serving, and that in consequence thereof amputation had been thought necessary on his return home. He had had a chancre eighteen years ago, followed by rash, and in India he had had ague, but in other respects his health had been always good. The history being somewhat unusual, and the sole of his left foot being evidently somewhat anæsthetic, I asked him to show me his other foot, and with astonishment found that his right great toe was also absent. The following history was then obtained; that when in India, eight years ago, his right foot began to be bad with a corn under the metatarso-phalangeal joint of his great toe; that having cut the corn, it began to discharge, became so sore as to incapacitate him for duty, and on his return to England the toe was removed in the Netley Hospital. About a year elapsed, and his left great toe was attacked in the same way, speedily got worse and was amputated. Then it was that he was told his right great toe had been lost by frost-bite, an explanation of his malady which to himself was wholly new, although he adopted it afterwards. In reality, the condition had in both instances begun with corns which ulcerated deeply, so that, as he said, "a probe could be passed right through" the metatarso-phalangeal joints.

In his gait there was nothing remarkable, nothing in fact which might not fully be accounted for by the loss of his toes; but on testing him with his eyes shut, he was decidedly unsteady. The knee-jerk was completely absent on both sides, the vasti, however, contracting normally on percussion. The soles of his feet were unquestionably anæsthetic. The pupils, which were small and not quite equal, reacted very sluggishly to light. For the last two or three years he has had pains in the legs. He called them "jumping pains," and said they had been so bad as to make him call out. Under the influence of hot fomentation to the foot, and the administration of iodide of potassium, the redness and œdema soon subsided, and he ceased to attend the hospital. I found him again on the 27th of February, 1883, when he told me that shortly after his last visit, or towards the end of January 1882, a corn formed under the fourth toe of his left foot, and that there followed a train of events which led to the removal of this toe in the Middlesex Hospital, he having failed to gain admission into St. Mary's. He was in the hospital for seven weeks, and was discharged well.

Some time in April 1882, a fortnight or so after he left the hospital, his left leg and foot began suddenly to swell as high as the knee. The swelling lasted for a week, and when it then subsided the ankle was left enlarged. This ankle has been swollen ever since, and there is now well-marked thickening, which is obviously due to bony enlargement of the lower end of the tibia and of the astragalus and os calcis. The ankle movements are tolerably free, and there is no bony crepitus. The ankle and foot are strangely widened, presenting very much the aspect of the foot of the patient who was shown by me at the International Medical Congress 1881,[1] and whose case has been more recently brought before the Clinical Society.[2]

The history of this joint attack so closely resembles that of the other case, in which both feet were affected, and unquestionably as symptoms or consequences of tabes dorsalis, that there can be little doubt of the ankle deformity being in this case also a true Charcot's arthropathy. The other phenomena remain the same.

There are few subjects at the present time of greater interest than the connection of such trophic changes as this case presented with central lesions of the spinal cord. Is the particular form of ulcer called "perforating ulcer" of the foot to be placed in the same category as the arthropathies which are not uncommon in the course of tabes dorsalis, or is it solely due to a peripheral neuritis which may sometimes precede, or even be a cause of, the special degenerative lesion of the spinal cord? Was this patient when he lost his big toes the subject of tabes dorsalis, or is the tabes which he now has but a later consequence of the peripheral disease to which his perforating ulcers were originally due? In the exhaustive paper on perforating ulcers which they brought before the Royal Medical and Chirurgical Society in 1879,[1] Mr. Savory and Mr. Butlin remarked that, "if called upon to express an opinion of the nature of the disease, we should say that the so-called ulcer is the result of pressure, or violence, or injury to structures, whose nutrition is impaired, or whose vitality is defective from disease or degeneration of the supplying nerves. In proof of this we would draw attention," they write, "to the fact that marked changes have been discovered in the nerves passing to the affected part in every case in which they have been thoroughly examined." Professor Ball and Dr. Thibierge, however, see in some cases, though not by any means in all, a more central cause than degeneration of the peripheral nerves, and in the paper read by them before the International Medical Congress in 1881, they come to the conclusion[2] that "perforating ulcer of the foot is unquestionably connected, in a tolerably large number of cases, with the earlier or later periods of tabes dorsalis," presumably, that is, with the *cord* degeneration which is peculiar to this disease. That there may be, however, other forms of "perforating ulcer" is acknowledged by these authors, and examples thereof have been recorded—to refer only to recent contributions—by Mr. Treves in a paper on "Perforating Ulcer of the Foot, and progressive Locomotor Ataxia," in the 'Lancet' of Oct. 21, 1882. He relates a case in which the original ulcer of one foot "made its appearance before any evidences of ataxia had developed;" and doubting, therefore, the causal relationship in that case between the ulcer and the disease of the spinal cord, he refers to three other cases under his observation during the last few years, where there were neither anæsthesia, nor undue sweatings of the foot, nor any evidence of nerve affection of any kind. He urges that "perforating ulcer," is a purely local affection, produced by purely local causes, which would act with increased vigour upon a part whose vitality is in any way impaired, but that

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[1] Vide 'Transactions,' vol. i. p. 124.

[2] 'British Medical Journal,' vol. i., 1883, p. 772.

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[1] 'Trans.' vol. lxii. p. 382.

[2] 'Trans.' vol. ii. p. 55.

that impairment is not of necessity dependent on disease or degeneration of the supplying nerves. In other words, he would not regard the ulcer as a trophic change.

There is, doubtless, no inherent reason why an ulcer of this particular kind should not be dependent on local causes only, set up by the long-continued and painful pressure of a corn on the soft parts beneath it, by consequent inflammation and suppuration, and by the deep burrowing of pus in the line of least resistance, so as ultimately to reach the bone. The association, however, of this form of ulcer with some degeneration of nervous structures is too frequent not to raise a suspicion that there is indeed some closer bond between the two. In the case with which this paper was opened there was in all probability nothing at the time when the patient lost his toes to suggest the existence of central disease, and five years went by before he began to have those jumping pains which M. Charcot and others have clearly recognised as being amongst the very earliest symptoms of true tabes. M. Charcot, it may be remembered, has even suggested that the first period of tabes dorsalis should be called the "period of lightning pains," because they "may of themselves alone compose the entire symptomatology of the disease." In Mr. Treves' case, also, a period of five years elapsed before his patient showed undoubted symptoms of tabetic degeneration of the spinal cord; and several other cases have been recorded where perforating ulcer was coincident with lightning pains, or even preceded their onset by a considerable interval of time.[1] Such observations as these invite the question whether perforating ulcer may not sometimes be an early and premonitory sign of a disease whose more telling symptoms are not yet developed, and whether the degenerative changes in the spinal cord may not even be an extension, or consequence, of those changes in the peripheral nerves which MM. Savory and Butlin assure us have been discovered in every case in which they have been thoroughly examined.

Grave pathological changes in the nerve-centres may, it is well known, be originated by extension of disease from peripheral lesions of nerves, or by long-continued irritation of nerve-trunks, as after amputation of a limb.[1] Several cases of the kind have been recorded; and the fact that perforating ulcer appears not unfrequently to be an early lesion in cases which ultimately show the true symptoms of tabes dorsalis suggests this important question, whether the degenerative changes in the spinal cord are not in such cases a direct consequence of the changes in the peripheral nerves? True, a weighty objection to this theory seems to present itself in the fact that the lesion of tabes dorsalis is almost strictly limited to the posterior parts of the spinal cord; but the objection loses weight when we call to mind that, as Savory and Butlin point out,[2] "the symptoms of nerve lesion in all cases point to affection of sensation and of nutrition, rarely to affection of motion; and never to affection of motion without affection of sensation or nutrition. In other words, the sensory and nutrient, or vaso-motor nerves, are at fault, the motor nerves generally unaffected." And, speaking of the microscopical changes, they go on to say: "It is of course impossible in the transverse section of a mixed nerve to decide whether any individual fibril is motor or sensory or nutrient (*i.e.* vaso-motor, or perhaps trophic). But, inasmuch as the fibrils which compose the sympathetic nerve are for the most part small, and either non-medullated or almost without medullary sheath, it may be supposed that nutrient fibrils in mixed nerves bear similar characters, and that a certain number, therefore, of the smallest fibrils seen in a transverse section are nutrient. . . . And although it does not appear to be absolutely true," that sensory fibrils may be distinguished from motor fibrils by their thinner medullary sheaths, "inasmuch as motor nerves contain thinly-medullated fibrils, and sensory nerves contain some thickly-medullated fibrils, there is generally so distinct a difference that we can recognise with ease a motor or sensory nerve. The fibrils of the former are larger, more thickly medullated, and more uniform in appearance. We believe, therefore, that the smallest fibrils of a mixed nerve are chiefly nutrient and sensory, and that the larger fibrils are chiefly motor. The histological characters of the nerves in the two cases examined by us strengthen this belief. Both are characterised by absence of, or damage to, or obliteration of, the small fibrils, either from defect, which is perhaps congenital, or from disease, whilst the larger fibrils remain apparently intact." The defects, therefore, of sensation and nutrition, which are often observed in cases of perforating ulcer, and of which latter the ulcer itself is one of the most striking symptoms, seem to these authors to be "due to absence or degeneration of the sensory and nutrient fibrils of the supplying nerves." And if it be true that these fibrils are especially picked out for damage, whether primarily from some unknown cause, or as the result of mechanical pressure or constant irritation of the more delicate constituents of a compound nerve, it does no violence either to physiology or pathology to assume that in its centripetal march the degeneration should involve those parts of the spinal cord which are in most immediate connection with the sensory roots. The essential degeneration of tabes dorsalis is limited to the sensory tracts of the spinal cord; as Leyden puts it, tabes is "eine Erkrankung der sensiblen, d. h. der centripetal leitenden Faserelemente," and there is neither more nor less difficulty in accounting for a degeneration being limited to one portion only of the spinal cord than to one set of fibres only of a nerve. And whether seated in the one part or the other, whether in spinal cord or in compound nerve, the degeneration will follow a true physiological—in this instance centripetal—march. Pathological investigation has yet to show us where that march begins, and I would urge a much more extensive inquiry into the morbid appearances of the peripheral nerves in all cases of tabes dorsalis which come to the post-mortem table. "In der Regel nimmt die Intensität der Rückenmarks-Erkrankung von unten nach oben ab und ist im Lendenmarke am intensivsten; doch giebt es auch Fälle, wo das Lendenmark relativ wenig, dagegen der mittlere Brusttheil, oder gar die Cervicalanschwellung viel stärker ergriffen sind."[1] What is the explanation of the rule that the lumbar portion of the spinal cord is most frequently and most severely affected with the degeneration of tabes ? Is it impossible that the explanation may sometimes be found in degeneration of those peripheral paths of the nervous system which may be far more exposed than are the central paths to unnatural and morbid influences ?

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[1] See a case recorded by Duplay and Morat, 'Arch. Gén. de Méd.' 1873, p. 268; one by M. V. Hanot, 'Arch. de Physiologie,' 1881, No. 1, p. 157; and one by Mr. F. A. Southam, 'British Medical Journal,' vol. i., 1883, p. 1222.

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[1] See Weir Mitchell's "Injuries of Nerves," and a recent case recorded by M. Charcot, 'Progr. Méd.' 1883, p. 157, *et seq.*; and Dr. Ferrier's paper on "Hemiplegic Muscular Atrophy of Peripheral Origin," 'BRAIN,' Vol. V. p. 521.

[2] *Op. cit.* p. 383.

At any rate we have these facts before us; that perforating ulcer is a symptom or consequence of some nutritive derangement of the affected part, and that in a goodly number of cases tabes dorsalis has been detected some time after this symptom of peripheral nerve-lesion existed. And it seems not altogether out of the question that in such cases the disease may have really begun at the peripheral parts of the general nervous system; that the nerve lesion was originally caused by the continued painful—often severely painful—pressure of the corns, by the protracted sensory disturbance giving rise to ultimate structural change, and that in the course of time—now shorter, now longer—the degeneration, travelling upwards, reached the spinal cord, and then, and only then, gave rise to the symptoms of tabes dorsalis. The question can only be solved by the careful observation of cases of perforating ulcers over a long period, that it may be known whether in any considerable number of them tabes dorsalis is ultimately developed, there having been originally no symptom of this central disease.

If such a sequence of events be clearly established, it will go a long way to support the view which, not unmindful of many objections, I have here ventured to put forward, that tabes dorsalis may in some cases have a peripheral beginning, and that a painful corn under the metatarso-phalangeal joint is a by no means trifling affection, little deserving of treatment. Present facts are suggestive; future observation can alone give conclusive answer to the question "May tabes dorsalis sometimes have a peripheral origin ?"

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[1] Leyden, 'Real-Encyclop.', Bd. xiii. p. 366, 1883.

NOTE ON THE PATELLAR KNEE-JERK.[1]

BY VICTOR HORSLEY, F.R.C.S., B.S. M.B. LOND.

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In view of the uncertainty at present existing with regard to the physiology of the so-called patellar reflex, I venture to record a few observations on the condition of this phenomenon when the subject of experiment is under the influence of nitrous oxide gas.

In 1881, while experimenting (on myself) with this gas for a different purpose, it occurred to me to contrast the conditions of the superficial and deep "reflexes," taking the plantar reflex as an example of the former, and the patellar phenomenon to illustrate the latter. Being aware of the fact that deep chloroform-narcosis abolishes all the "reflexes," superficial and deep, and yet that with ether-narcosis, however deep, ankle-clonus frequently appears, I was scarcely surprised to find that the knee-jerk persisted in the deepest anæsthesia from nitrous oxide, while the superficial reflexes all disappeared. This distinctive difference between the two groups of "reflexes" was so striking, as to merit further observation, of which the following is a brief summary.

The necessity of estimating exactly the degree of consciousness in dealing with an anæsthetic reagent, such as nitrous oxide, is obvious when the statements of patients (who have been operated upon while under its influence) are taken into account. To avoid the possibility of error in stating the depth of narcosis, only the result of experiments (fifteen in number) made on myself is here stated, but the facts were verified by observations made on other subjects.

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[1] I desire here to express my obligations to my friends, especially to Mr. Henry Maudsley and Mr. Halliburton, for kind help in making the above observations.

In all cases the anæsthesia produced was complete; after the usual symptoms attending the commencement of the administration of the pure gas were fully marked, absolute unconsciousness followed in from 90 to 120 seconds.

The anæsthesia was pushed until rigidity and sometimes cyanosis resulted. The recovery of consciousness was very frequently attended with considerable muscular spasm and semi-co-ordinated convulsive struggles and excitement. The "reflexes" were examined at regular intervals of five seconds from the commencement of the experiment to the return of consciousness; the sole of the foot being pricked with a pin, for the plantar reflex, while the opposite patellar tendon was struck by a stethoscope or the hand. (The knee-jerk being normally very well marked, there was no opportunity for error of observation in eliciting the phenomenon, and the plantar twitch was also very active.)

Result.—In the deepest narcosis the condition of the reflexes was as follows:

Superficial.—Plantar and conjunctival abolished.

Deep.—Knee-jerk persistent.

Thus it is seen that although the whole central nervous system, viz. the encephalon and spinal-cord, was poisoned by the gas, the knee-jerk still continued active.

Taking this fact in connection with the short time expended in the knee-jerk contraction, the conclusion is unavoidable that the shortening of the rectus is due to local irritation of the muscle from sudden elongation, the integrity of the normal tonus being maintained. It seems scarcely necessary to refer to this latter point, but in an admirable paper on this subject,[1] Dr. Prevost has shown, that in rabbits, anæmia of the lumbar enlargement of the spinal-cord (produced by compression of the abdominal aorta) is followed by the simultaneous (?) disappearance of the deep and superficial "reflexes." It would appear, however, that in nitrous oxide we have a reagent with which it is possible to perform the crucial experiment of suppressing the superficial reflexes without abolishing muscle tonus. With regard to the different results obtained by using different anæsthetic agents, the following conclusions may be drawn from the researches of Eulenburg,[1] also confirmed by Prevost's experiments with ether, &c., viz. (1) that the deep "reflexes" are only abolished by the denser anæsthetic reagents, such as chloroform, &c., and are exaggerated by the more volatile reagents, such as ether, &c., but that both superficial and deep "reflex" action is completely abrogated in the asphyxial state; and (2) that in the latter condition, whether induced mechanically, or by drugs such as nitrous oxide gas, the superficial disappear before the deep "reflexes."

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[1] Published in the 'Revue Médicale de la Suisse Romande,' Jan., Feb., March 1881.

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[1] 'Centralblatt für Med. Wissensch.' No. 6, 1881.

Clinical Cases.

A CASE OF EPILEPSY—AURA OF TRUE VERTIGO WITH AUDITORY SENSATIONS—VAGUE COMPLEX REMINISCENCES—EMOTION OF HELPLESSNESS—SPASM PRIMARILY LEFT-SIDED.

BY CHARLES MERCIER, M.B. LOND., F.R.C.S.

Eliza Ann A., æt. 49, married; three children living; three dead. No miscarriages.

No family history of neurosis, save that a niece of patient's had puerperal insanity. Patient persistently attributes her illness to "change of life." Her health was good until Christmas, 1881. At that time, while walking in the street, she had the first of a series of attacks, which have recurred at intervals ever since. In this attack she felt giddy, reeled about, and fell. She felt "as though she was going to lose her senses." She had to catch hold of something, and then fell down. She felt as if she was going round. This feeling of going round was, she states in answer to questions, "like a dreadful giddiness." When asked in which direction she went round, she alters her position, and appears to make a careful effort at remembrance. She then says positively that she seemed to go round from left to right. She is very positive that this was the direction in which she appeared to turn, and she adheres to this statement when repeatedly cross-questioned at different times. During the onset of the attack she had other feelings. She is not clear as to whether these feelings existed at the same time as the vertigo, but it would appear as if sometimes these feelings and sometimes the vertigo was the preponderating sensation at the outset of a fit. The feelings she describes as follows: it is "as if I was going a long distance;" "as if I was going further off;" "as if I was going right out I don't know where;" "once I felt as if I was going down into a pit;" on another occasion the feeling was "as if there was something in front of me and it broke off, so that I could not get through it; I tried and tried, and could not get through it." On another occasion this feeling "came on as I was taking the kettle off the fire, and I had to put the kettle down and run out of the room and catch hold of the banisters." She states that the attacks are accompanied by strange feelings in her ears. She volunteers the statement without being questioned, " that they are not in her head so much as in her ears. It is, she says, " as if something was going right through her ears." On one occasion she heard a loud bang in her ears, " as if a tea-tray had been struck a violent blow." (She has no defect of hearing with either ear.)

After she fell down in an attack she " lost her senses altogether;" but she subsequently modifies this by saying that she could hear that people were talking around her, and could distinguish what they said. During many attacks she was conscious throughout, and knew that she was " like this " (here she imitates most accurately the facial movements of a convulsion beginning in the left side of the mouth and affecting the eyes). She states, without questioning or prompting of any kind, that the left eye worked much more than the right, and she imitates the characteristic twitching with remarkable fidelity. She goes on to say that her arms also would work about; and here she imitates fairly well the movements of bilateral clonic convulsion. The legs, she says, were not affected so badly as the arms; but sometimes they would shake about, especially the left leg (*n. q.*), and here she imitates the movement of clonic convulsion in the left leg. She occasionally—but not, she thinks, during a fit—has a feeling going down the arm; she particularises the left arm, but says, in answer to questions, that she feels it in both. She is certain that it goes *down* the arm, sometimes beginning in the shoulder, and sometimes in the elbow. She says it is like a powder going down the arm, and at the end a feeling comes at the tips of the fingers like pins and needles in them. After a fit she feels " very bad all over," and " as if her feet were in dough."

To direct leading questions, she says that she never felt angry during nor after a fit. She is right-handed.

Remarks.—The foregoing record is a contribution to our knowledge of a class of very obscure and recondite cases, to which attention was first directed by Dr. Hughlings-Jackson in Part X. of 'BRAIN,' and resembles in many respects a case that I shortly afterwards contributed to these pages in illustration of his views. From the point of view posited in Dr. Jackson's article, the present case is very interesting and important.

The main conclusions arrived at in the article referred to—conclusions which were, however, avowedly tentative and provisional—were: (1.) That all " warnings " of fits, in other words, all mental states occurring at the outset of a fit, may be divided into two classes, according as they are developments, however crude, of objective or of subjective sensations; by objective sensations being understood those feelings which normally occur during reaction of the organism on the environment; and by subjective sensations, those which occur during action of the environment on the organism. (2.) The left cerebral hemisphere is that which represents the most objective movements—those movements which are concerned in primarily operating on the environment; and is that during action of which the objective sensations arise. To this order belong the movements concerned in orientation, whose disorder is accompanied by vertigo; movements of the right hand, which is the leading member in acts on the environment; the movements of speech, which is "a process by which are symbolised relations of things in, or as if in, the environment; that is, things considered objectively, and whose disorder is aphasio." (3.) The righ themisphere, on the other hand, is active during sensations of the subjective order, and its activity gives rise to the answering movements. It is active during sensations of smell and taste, during hunger, one of the most subjective of all sensations; and its activity gives rise to the answering movements of mastication, deglutition, spitting, &c. Feelings of colour, the subjective side of the function of vision and of hearing, are also connected with the action of the right hemisphere.

If these suppositions are correct, we must expect to find that when vertigo occurs at the outset of a fit, or when aphasia occurs among the sequelæ, or when an emotion belonging to the objective series is aroused, the accompanying spasm will be primarily right-sided, all these occurrences being manifestations of the action of the same hemisphere—the left. On the other hand, "warnings" of coloured vision, of smell, of noises in the ears of the epigastric sensation, together with emotions of the subjective order, should be accompanied by left-sided spasm, since this occurs from, as those occur during, action of the right hemisphere.

The "complex vague reminiscences," or "dreamy states," such as occurred in the foregoing case, are, in Dr. Hughlings-Jackson's experience, mostly associated with warnings of the "subjective" order, and with left-sided spasm; and the warnings in such cases are not only of the subjective order, but are of the most strongly pronounced subjective character;—such warnings as those of smell, of the epigastric sensation, and presumably of taste. It is rare in his experience for the dreamy state to attend warnings of coloured vision and noises in the ear, and he had at the time of writing (1880) taken notes of only one case in which this state occurred with the warning of definite or true vertigo. Although Dr. Jackson states that this dreamy state is usually associated with spasm beginning on the left side, it would appear that this opinion is merely the epitome of his experience, experience which, considering the rarity of the cases, may easily prove exceptional; and in any case the validity of his inferences is not in the least impaired by the occurrence of these states in association with the objective warning of vertigo, as in the present case, and as in the previous case that I published. I believe that it is not the occurrence, but the character of the reminiscence, which is associated with spasm of one or other side.

Turning now to the case here recorded, we find that in some respects it is in harmony with Dr. Jackson's reasonings, in some respects it is anomalous with regard to them, and in some respects it is exceptional to his experience. It is exceptional to his experience, as already pointed out, in the association of the "dreamy state" with true vertigo and noises in the ear. It is at variance with his conclusions in so much that vertigo, a feeling belonging to the objective order, is associated at the onset of the fit, not only with noise in the ears, a feeling of the subjective order, but with spasm primarily left-sided, that is, with action of the right hemisphere. Again, the vertigo was of such a character that the patient felt as if she herself was turning from left to right, which is equivalent to the displacement of objects from right to left; which is in harmony with the commencement of the spasm on the left side indeed, but is opposed to the hypothesis that the vertigo occurred during discharge of the left hemisphere.[1]

On the other hand, the points as to which this case harmonises with Dr. Jackson's expectations are much more numerous and are very important. The "dreamy state," or reminiscence, did, it is true, accompany vertigo, a feeling of the objective class; but then it is a striking fact, and one which strongly corroborates Dr. Jackson's notions, that *this reminiscence was itself of a markedly objective character.* Vertigo is a motor disorder—a disorder of a group of highly representative movements or motor arrangements; and, correspondingly, every one of the vague complex mental states which in this case occurred together with, or in connection with, the vertigo, was a reminiscence of *movement.* "As if I was going a long distance," "as if I was going further off," "as if I was going right out I don't know where;" these are the patient's expressions; and in every case the idea is one of movement, not initiated by, or connected with, anything happening in the environment, but of movement spontaneously initiated by the organism.

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[1] After the above paragraph was in the press, a more careful perusal of Dr. Hughlings-Jackson's article convinced me that I had greatly exaggerated the discrepancies between his conclusions and the facts of this case. The vertigo which he believes to be accompanied by right-sided spasm, and to signify discharge of the left hemisphere, is that vertigo in which there are *apparent movements of objects to one side* (the italics are Dr. Jackson's). Vertigo such as occurred in the present case is explicitly distinguished by him from the form of vertigo which he believes to accompany right-sided spasm, and to be so rarely associated with the dreamy state. The distinction is so clearly and repeatedly stated that I am at a loss to know how I can have overlooked it. I can only offer the explanation given by Dr. Johnson in a similar case,—"pure carelessness."

These reminiscences, of which the above is the cognitive aspect, are accompanied by feeling; and the feeling, which is strongly pronounced, is of a strictly corresponding order—the objective order. Dr. Jackson considers anger an emotion of the objective order, and contrasts it with fear, which he gives as an example of a subjective feeling; but it seems to me that anger as well as fear must be considered subjective in the sense in which he uses this term, of "answering to what is physically the effect of the environment on the organism." Anger, no more than fear, can arise without some circumstance in the environment first acting to elicit it. And not only are both anger and fear primarily initiated by the action of some agent in the environment upon the organism, but in both an equivalent reaction takes place. It is true that the reaction in one case takes the form of counteraction, while in the other it takes the form of avoidance; but it seems to me that a dog running away from a bigger dog displays as energetic a reaction as the same dog fighting a smaller one; and the accident of the size of the object cannot surely make a difference in the *order* of the feeling that it evokes. Feelings of the objective order, properly so called, appear to me to be not those which accompany movements that are reactions in any proximate sense on the environment, but those which accompany actions primarily, spontaneously, and *proprio motu* initiated by the organism. A simple example of such feelings is Effort, and among the more abstract and highly evolved feelings of the same order are the emotions of Power and Helplessness. The latter is the feeling which accompanied the other manifestations of irregular objective activity experienced in the case under consideration. The patient states that she had a feeling "as if there was something in front of me, and . . . I tried and tried, and couldn't get through it." Evidently here was a vague but massive feeling of helplessness, a feeling which is, as has been said, of strictly objective character.

Not only was the cognitive aspect of the reminiscences of a markedly objective character; not only was the accompanying emotion of an order strictly objective; but the vertigo, which is always an objective manifestation, had in this case the character of ultra-objectivity. For the giddiness was not an experience of things going round, not, that is, a sensation cognised as being initiated by things in the environment; but it was described by the patient to be as if *she herself* were going round; that is to say, it accompanied the activity of motor processes that represent spontaneous movements. It is remarkable that in the only case that Dr. Hughlings-Jackson adduces as experiencing the dreamy state with definite vertigo, this dreamy state was one conspicuously objective in the sense here used. It was a feeling purely though crudely motor, —a feeling of being in some other place—several places, not any particular place—that is to say, it had not enough of the subjective or environmentally-initiated element to give it even the vaguest localisation.

Lastly, my patient has, with the other mental accompaniments of spasm, auditory sensations more or less definite, or at any rate feelings referred to the ears, and these feelings belong not to the objective but to the subjective class. Knowing how closely the ear is associated with the function of orientation, we must not be surprised at this, although in some respects it is in conflict with the suppositions made.

To summarise the facts of this case. There occur at the outset of the attack (1) true vertigo, a sensation always of the objective order, and in this case ultra-objective; (2) a vague representation of spontaneous movement,—a cognition of the objective order; (3) an emotion of helplessness,—also of superlatively objective nature; (4) an absence of all emotions of the subjective class; (5) auditory sensations, which, though primarily of the subjective order, are yet, regard being had to the vertigo, not altogether anomalous; and (6) these conditions, all connected together by one underlying similarity into a coherent group, are associated (in a right-handed person) with spasm primarily left-sided, an association altogether exceptional, although not perhaps altogether inexplicable. There is at least a definite preponderance of the spasm on *one* side, and although this side happens to be the wrong side, according to the hypothesis, yet in occurrences of such extreme complexity, we must be thankful for any definite association of them, even if it make matters at first more puzzling than before.

It should be stated that, as will be seen from the foregoing, the connotations attached herein to the terms "objective" and "subjective" are not absolutely identical with those used by Dr. Jackson, although they are closely similar.

A CASE OF HYSTERICAL (?) RAPID BREATHING IN A YOUTH.

NOTES BY M. W. H. RUSSELL,

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CHAS. E., aged 19, single, paper-hanger, admitted into Duncan Ward on December 27, 1882, under the care of Dr. Fox.

There is no history of any nervous disorder in the family; no history of phthisis, and none of rheumatism or chorea.

Patient had measles, scarlet fever, and pneumonia at the age of 3 years; was not ill again until 13 years old, when he had "low fever." Never had fits. He recovered well from the low fever, and with the exception of some shortness of breath (panting after any unwonted exertion), which appears to have first manifested itself about this time, he remained fairly well until two years ago.

Two years ago the shortness of breath became more marked, and he attended as an out-patient at this hospital, and at a dispensary in the town, without deriving much benefit. At this time the shortness of breath was not attended by any unusual symptoms.

It is only during the past six months that the dyspnœa has acquired its present form. For that time he has been entirely precluded from work, on account of attacks of extremely laboured breathing, complicated with twitchings of face-muscles and spasmodic jerkings of limbs, and other nervous phenomena. Any excitement greatly aggravates the symptoms. At the end of attacks, patient for the last six months has been noticed to "bark like a dog."

Patient is a thin, pale, emaciated, dark-haired youth. He is in a most distressing condition. He has paroxysms, occurring at intervals of about 15 seconds, of forced breathing, in which he sets all his extraordinary respiratory muscles to work. He cannot lie down, and though much exhausted when the paroxysm comes on, he leans forward, fixing his hands on the bed, so as to give a *point d'appui* for his pectoral muscles, which, together with all muscles which can aid respiration, are brought into requisition. The shoulders move up and down, the alæ nasi are hard at work, the mouth is held half open.

Each paroxysm consists of about 20 to 30 forced and hissing inspirations; each successive inspiration is deeper than its immediate predecessor, the last one being apparently the acme of his inspiratory power. The series is ended up by a peculiar rough, barking noise, several times repeated, as though he were attempting to hawk up some very viscid secretion in his large tubes. Nothing is however expectorated. The noise was mistaken by a physician in the ward for a carpenter sawing wood. The paroxysms are not followed by any period of apnœa, as he breathes quite tranquilly and evenly in the intervals, which are rarely longer than 15 seconds in duration. He has a peculiar furtive, helpless and alarmed expression of face, more marked during the actual paroxysm. When spoken to, he exercises considerable control over himself, and checks the seizures sufficiently to be able to answer clearly and intelligently any questions; but when not actually speaking, his distressing condition is immediately re-estalished.

Chest.—Long and narrow, walls emaciated, lower spaces very concave; expansion movements almost entirely eclipsed by elevation movements, which are much exaggerated. Area of pulmonary resonance is increased in all directions, and the note itself is fuller than usual. Full resonance over upper sternum. Cardiac dulness obliterated, and upper level of hepatic dulness depressed about 1½ inch.

All over chest the loud hissing laryngeal sounds are heard conducted, masking all other sounds during the attack; during the intervals the normal breath-sounds are obscured by sonoro-sibilant râles. Expiration is much prolonged.

Heart.—Apex felt feebly beating in normal situation. Apex sounds muffled. Basic sounds very distant. No bruit heard at base or apex.

Abdomen.—Liver felt 1 inch below costal margin, spleen not felt, no dulness made out.

Urine.—Acid, s. g. 1020; no albumen, no sugar. T. 99·6: P. 76.

Ordered a mixture containing: Liq. sod. arsen. ♏iv; chloral hydr. gr. xx; potass. bromidi gr. xx aq. ℥i, *ter die.*

The pulse is not found to vary materially during the respiratory explosions. It is occasionally a little accelerated.

Dec. 28th.—Patient remained in much the same condition all through yesterday afternoon. Towards evening, after the second dose of his medicine, he began to get drowsy and early fell asleep. He slept well and soundly, and breathed quite easily and quietly. Immediately on waking, the peculiar rhythm of respiration was re-established, but not with such painful intensity, the intervals being a little longer (20 seconds), and the breathing itself less forced. T. 98·2: P. 92. The sucking in of lower intercostal spaces is very marked during the laboured breathing. Patient exhibits slight choreic symptoms when watched; the brows are knit, the mouth pursed, the eyelids twitched. Bowels open. E. T. 99. Patient remains same. Sleepy.

Dec. 29th.—T. 98·8: P. 96, of fair quality.

Patient has had another good night and has since been dozing. Is perfectly quiet when asleep. Average length of intervals to-day 20 seconds; the paroxysms themselves consist of 15 forced inspirations. Patient has greatly recovered from his exhaustion. He can ward off seizures longer when talking. The barking is less harsh than it was.

E. T. 98·6: P. 72. Has dozed a good deal during the day. Is easily awakened, and immediately the respiratory phenomena recur. Each paroxysm now consists of ten rapid forced inspirations on an ascending scale, i.e. each one increasing in depth. The whole are performed in about 19 seconds.

The barking is to-day very occasionally omitted.

Dec. 30th.—T. 98. Patient is picking up well in his general health. The intervals are not much prolonged, still averaging about 25 to 30 seconds, but the respiratory efforts are less distressing and less panting.

Ordered to continue mixture, with the addition of Liquor atrophiæ sulph. ♏ 1½ to each dose.

Dec. 31st.—T. 98·6. Complains of throat feeling dry. Pupils are slightly dilated. E. T. 98·8.

Jan. 1st.—T. 99. Has had a very good night. Takes food well. Fits continue to diminish in severity, and bark is only once, twice, or at most thrice repeated after each. It is also less harsh. E. T. 98.

Jan. 2nd.—T. 98. Improvement maintained, but very slow. Seems fairly comfortable. Whenever awake, patient does not escape. The fits come on every 20 or 30 seconds, but are now reduced to four or five panting respirations and a bark. Sleeps and eats well. E. T. 98.

Jan. 3rd.—T. 98: P. 80. Small and feeble. The pulse is to-day found to slow slightly at the commencement of each attack. These remain of only moderate severity, but the intervals are most irregular, and are often as short as 5 or 6 seconds. Barking less. Throat very dry.

Mixture to be taken only twice a day.

Jan. 4th.—T. 98. Patient seems disturbed this morning.

Breathing more jerky; more barking. Paroxysms increased again to 8 or 10 breaths.

Jan. 5th.—T. 98·6. More excited. Intervals only 6 seconds. Paroxysms 15 or 20 breaths. Slept well.

Ordered Santonin, gr. iii; calomel gr. iii; to be taken at once, and to be followed by ol. ricini ℥ss. *cras mane.*

Jan. 6th.—T. 98. Slightly quieter. Bowels open. No worms expelled.

Ordered, Liq. arsen. ♏ij; pot. brom. gr. xv; chloral hyd. gr. xv, t. d. s.

Jan. 7th.—Much quieter again. Paroxysms less frequent. Has intervals of 4 or 5 minutes. Patient says that as the attacks become less frequent, his breathing feels more oppressed.

Jan. 8th.—Paroxysms rather longer (20 or 30 breaths). More barking; choreic twitching more marked.

Jan. 9th.—Quieter. Mixture omitted.

Jan. 10th.—Much more unruly again; scarcely ever free from forced breathing. Feels giddy after fits. Bowels open.

Ordered Zinci valerian. gr. vi, in pil. t. d. s.

Jan. 11th.—Distinctly worse. Intervals reduced to 6 seconds. Fits altered in character, consists of about 30 inspirations, culminating in a hiccough, after which one bark. At times the intervals are abolished, and the forced breathing is kept up for over a minute.

Replaced on Pot. brom., chloral and atropia.

E. T. 98·6. Ophthalmoscopic examination revealed nothing abnormal in the fundus of either eye. It was noticed that when the patient's attention was concentrated upon the ophthalmoscope, the respiratory spasms were held in check.

Jan. 14th.—Very much quieter.

Jan. 28th.—Been gradually improving since last note.

Feb. 10th.—Still slowly improving.

Feb. 28th.—Discharged. As soon as the medicine is stopped, the fits return.

March 20th.—Came as out-patient. Has been taking medicine regularly since discharge, and has enjoyed almost entire freedom from symptoms.

April 2nd.—Came again as out-patient. Still finds old symptoms return as soon as medicine is withheld. Is very anæmic. Has a few very mild attacks still daily.

August 10th.—The patient presented himself again to-day. His symptoms still persist, becoming much worse if the medicine is left off for more than two days. Whilst taking the medicine, they are kept more or less in abeyance.

CASE OF PRIMARY LATERAL SCLEROSIS OR SPASMODIC TABES.

BY JOHN HOPKINS, F.R.C.S., ENG.

Medical Superintendent, Cleveland Street Sick Asylum, W.

J. M., aged 21, porter, was admitted August 14th, 1881. Father and mother living, and stated to be in good health. All brothers and sisters, thirteen in number, died under four years of age, two of them of smallpox.

Patient had fits when a child. Never had venereal disease, smallpox, or any illness till twelve months ago, when he got wet, and within a week found his legs becoming weak and dragging, and his gait tottering; he improved a little, but the weakness and tottering never left him entirely.

The following was the condition on admission. Patient is fat but pasty-looking, and feels in perfect health. No signs of congenital syphilis. Gait peculiar, rolls from side to side, and as each limb is brought forward it jerks irregularly, and when put down stiffens and trembles. No difficulty in standing with eyes closed. On tip-toe the legs tremble. Percussion of the patellar tendon causes not only heightened patellar reflex, but trembling of the limb. Ankle clonus marked. Slight loss of sensation in the sole of the right foot (?) No wasting of muscles. Arms not affected. No symptoms referable to special senses, tongue, stomach or bladder. Fundus oculi normal. Heart and lungs free from signs of organic lesion. Urine clear and free from albumen.

Patient for some months got up daily, ate and slept well, and expressed himself as feeling perfectly well, but walking became increasingly difficult, and all the symptoms aggravated. Passive elevation of the knee from the bed, or attempt at producing ankle clonus threw, first the one, then both limbs into violent trembling. For a time crutches were used, but had to be abandoned. On February 1st, 1882, it was noticed by the patient when he tried to stand, that there was scarcely any feeling in the soles of the feet; the ground, which simply felt warm to the feet, seemed to be sinking under them. On testing them, the prick of a pin was readily felt. Rigid flexion was noticed to come on for the first time on May 16th. It first occurred in the right lower limb, but in a few days affected the left also. The limbs could not then be extended passively. The spasm was not permanent; at times they could be extended voluntarily. On May 19th the urine was alkaline and purulent, and bed sores had formed over the sacrum and trochanters. A night's rest in a sitting posture gave a gangrenous look to the whole nates. However they recovered again. Wasting of the muscles at the front and back of the legs was noted then for the first time. On June 15th, patient complained of stiffness of the upper limbs. On examination they yielded very slowly to passive movement, both in flexion and extension, but there was no spasm that could not be overcome. There was no pain, tenderness, heightened reflex, or clonus in them.

The bed sores penetrated deeply to the bones through thick pads of fat. During the last week the temperature steadily rose, with remissions, accompanied by rigors and sweats, to 105·8° F. This was on July 12th: on the same day, the bed sore over the right trochanter bled freely. The next day the patient died. Temperature normal an hour before death.

Autopsy.—Body fat; lower limbs wasted. Serous cavities, heart, lungs, intestinal tract, liver spleen and kidneys normal. Rugæ of bladder congested, and trigone covered with mucus.

Brain and membranes, spinal canal and dura mater healthy.

The spinal cord presented marked gelatinous degeneration in the lateral columns which extended from the tip upwards into the upper portion of the dorsal region. The degeneration extended higher in the right side, and at a point above any visible sclerosis the column was congested, being in marked contrast to the rest of the white matter. In the lumbar enlargement the degeneration extended forwards into the anterior column and in front of this the white matter was greyer and firmer than normal. In one spot the anterior columns next the anterior median fissure were gelatinous. Above the lumbar enlargement the sclerosis did not invade the anterior columns to the same extent, and higher still the lateral columns were only in part affected. In the upper part of the dorsal region the gelatinous degeneration was visible as a thin band, just in front of the posterior cornu and parallel with it. After hardening in chromic acid the parts described above as sclerosed were stained brown, the tint varying with the degree of degeneration. Besides, the central grey matter was also stained in a similar manner in certain parts. In the mid-dorsal region and below this (except in the largest part of the lumbar enlargement), the grey matter was throughout darker than at the upper part of the cord. Above the mid-dorsal region the brown staining invaded less and less of the grey matter, the anterior cornua being free from it, while the posterior half of the grey mass was stained. Higher still, at a point where the sclerosis in the lateral columns was shown by a thin wedge-shaped tract on the right side, and a thin band on the left, the grey matter was very little discoloured.

Microscopic examination showed the lateral columns and neighbouring part of the anterior to be nearly devoid of nerve tubes at the lower end of the cord, and the anterior columns to contain a considerable quantity of connective tissue between the nerve tubes. In the dorsal region the lateral columns contained some nerve tubes, which were found in increasing numbers as the examination was extended upwards. The most sclerosed part of the column seemed to be not directly in contact with the posterior cornu. In the mid-dorsal region the anterior columns were comparatively free from connective tissue. The large cells of the anterior cornua were much atrophied in the lower half of the cord, except in one section taken from the lumbar enlargement; in this the cells appeared normal in size, and number; but a little lower where the cells were in three pretty well defined groups, a small central group, a larger antero-lateral and a large postero-lateral group, the two first groups were markedly atrophied on the right side.

Remarks.—In 'The Localization of Cerebral and Spinal Diseases,'[1] Charcot, in attributing the symptoms of spasmodic tabes to primary sclerosis of the lateral columns, says "the hypothesis at the present time has received no verification."[2] Although in the above case the sclerosis extended beyond the lateral columns at the lower end of the cord, the symptoms, taken together with the extent and degree of sclerosis of these columns found post mortem, make it more than probable that this was a case of primary ascending sclerosis of the lateral columns.

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[1] New Sydenham Society's Translation, p. 304.

[2] Dr. Dreschfeld has published a case since Charcot published this: *vide* British Med. Journ. 1881.

ELECTRO-THERAPEUTICAL NOTES.

BY WALTER G. SMITH, M.D. DUBLIN, F.K.Q.C.P.I.

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NOTWITHSTANDING the abundant evidence which has now accumulated in proof of the palliative and curative powers of electricity in the treatment of neuralgic affections, it must be allowed that its use is not yet so general as its undeniable claims to consideration justify. No consulting room can be said to be properly appointed that is not provided with a suitable induction apparatus, and a constant battery and accessories, *kept in good order*, and it is a sign of imperfect acquaintance with the teachings of modern therapeutics, if the medical practitioner neglect to avail himself of the resources which the legitimate use of electricity places at his command. I have no hesitation in expressing my opinion that in most cases of persistent or recurrent neuralgia, it is worth while to try the effects of a gentle galvanic or faradic current. When cautiously used, no ill-effects will ensue, and in many instances the success is so immediate and unquestionable as to be alike agreeable to the patient and gratifying to the physician.

Contrasting strongly with the important data to be gathered in affections of the motor apparatus, I am not aware of any observations tending to establish the diagnostic value of electricity in sensory affections, e.g. in distinguishing a central from a peripheral lesion; or, in framing a prognosis as to the mildness or gravity of a case.

In regard to the method of application of the current, it was for a time customary, and some writers still speak with confidence as to the relative merits of the so-called ascending or descending current, i.e. primary importance is attached to the direction in which the current is supposed to flow. This is a point which has occasioned much vain controversy and given rise to no little confusion of ideas, and to many idle and pseudo-scientific speculations. Clinical observers arrived at discordant results, or came to the conclusion that it was immaterial whether the direct or inverse current were employed. But we have learned to regard the matter from another point of view, and now recognise the importance of studying the "polar" effects of the current, i.e. the local influence exercised immediately beneath, and in the neighbourhood of either electrode placed over the desired spot, the other electrode resting on an "indifferent" locality in the median plane of the body.

Electro-therapeutical science is indebted to the recent investigations of Erb, and of Waller and De Watteville, confirming and extending our knowledge of the polar effects of electric currents, which have done much to elucidate a difficult problem in physiology, and to dissipate a cloud of perplexities and apparent contradictions which obscured the rational application of electro-therapeutical methods. It should be clearly understood that the dispute as to the relative effects of the "polar" (uni-polar) and "direction" (bi-polar) methods is really a verbal one, and that there is no essential difference between them, both resting upon the same fundamental principle.

In the field of *diagnosis* of motor affections, we have gained a good deal in exactitude of knowledge, and it is to electro-physiology that we look for correct methods and ways of procedure, and for the destruction of erroneous views. But while we fully recognise that electro-therapy cannot dare to emancipate itself from electro-physiology on account of the practical insufficiency of existing physiological data, we must face the fact that a rational system of electro-therapeutics is still to be waited for, and we must be content for the nonce to let it root itself in the soil of clinical experience. A system of therapeutics based upon the theory of electrotonus, for example, rests upon an unsubstantial foundation.

That brilliant cures in many neuralgias can be obtained by electric currents employed in very different modes, and that in the same disease similar successes can be got by physiologically opposed methods is undeniable, and in illustration of this I will cite a personal observation of Rossbach. For many years Professor Rossbach had been a firm believer in the soothing action of the anode. He, unfortunately, became the subject of a very severe sciatica, and in the course of treatment of this affection by electricity, he was convinced, in the clearest way, that the pain was relieved, not only when the anode was placed upon the painful points, but even much more quickly and decidedly, when the kathode was placed upon the same points and voltaic alternatives employed. In another case, one of agonising facial neuralgia in an old woman, he perseveringly applied the anode of the galvanic current for a long time without the slightest benefit, whereas the pain was relieved by the faradic brush in two short sittings. ('Lehrbuch der physikalischen Heilmethoden,' Dr. M. J. Rossbach.) Cf. abstracts of 17 cases in Erb's 'Elektrotherapie,' pp. 493–497. In conclusion I will adduce brief notes of some cases exemplifying the striking and permanent utility of electricity in controlling neuralgic conditions. Three of these cases occurred in the persons of medical men, two of whom wrote to me an account of the benefit they had experienced from the treatment.

Case 1.—Mr. J. C., aged 21 years, subject for several years in summer to attacks of neuralgia of the right side of the face. In June 1878, he was seized with severe pain over the right side of his face, especially about the upper and lower jaw. The pain came on in paroxysms 3 or 4 times a day, each paroxysm lasting about an hour. Exposure to a draught, or the act of drinking a cold liquid, would provoke an attack. The galvanic current (4–8 Leclanché cells) was applied for a few minutes; + pole over the painful spots, and − pole lower down and beneath the angle of the jaw. Immediate relief was obtained, and next day the current was again applied as some pain lingered about the orbital region. From the day following he was entirely free from pain, and has never, to my knowledge, had a relapse.

Case 2.—In March 1878, a young gentleman, Mr. B., consulted me for a violent attack of neuralgia in the right eye, of 14 days' standing. The pain shot through the ball of the eye constantly, causing lachrymation, and extended to the supra-orbital region.

He had taken quinine freely, and morphia had been injected subcutaneously over the eye, but without any benefit.

The continuous current was directed over the seat of pain, and, *after the first application*, the pain completely disappeared and did not return.

Case 3.—Dr. W., of middle age, subject since 19 years old to attacks of pain about the right hip and down the leg, subsequent to a fall. He has been laid up for 3 weeks at a time with sciatica, and ascribed his last attack to bathing. The current from 12 cells was applied; one electrode over the lumbar vertebræ, the other down the back of the thigh. Good effects were at once apparent, and the current was used some 6 or 7 times. He felt well and comfortable the whole day after the electrical treatment, was enabled to face cold wet weather without fresh pain being provoked, and was so convinced of the beneficial effects of electricity in his own person, that he procured a Leclanché battery, and on future occasions was accustomed to check the first threatenings of return of the pain by a timely application of the current.

CASE 4.—Reverend J. C. was on the point of starting for the S. of Ireland, when he was suddenly seized with agonising pain in the loins and hips. Hypodermic injections of morphia and other sedatives proved of little use. When I first saw him, at Dr. Head's request, he had been several days ill, was lying on the bed, sweating with pain, and the least stir caused such distress that he had not dared to undress himself, and every act of respiration sent a twinge of pain across him. He had not slept for some nights. The effects of the continuous current applied to the painful parts was very striking. After a few minutes he ventured to turn in bed, was then able to undress and lie comfortably in bed, and in 3 or 4 days he left for Cork, and has since had no return of the pain.

Reviews and Notices of Books.

History of the Criminal Law of England.—By Sir JAMES FITZJAMES STEPHEN, K.C.S.I., D.C.L., a Judge of the High Court of Justice, Queen's Bench Division, London. Macmillan, 1883.

THIS important work contains a great chapter of especial interest to medical men, on "The Relation of Madness to Crime," so different in tone and quality to anything which has before been written by anyone of the author's profession that it stands, and cannot fail to remain, as a mindmark from which all further advance in the solution of one of the most difficult problems of legal and medical and social science must be measured. "The plea of insanity is unquestionably the most difficult corner of the Criminal Law" once said to us a great judge, than whom no one who ever thought on the subject was more competent to form an opinion; and a difficult corner we fear it will ever remain, from the inherent obscurity and intricacy of the questions involved. And this new contribution to its literature is far from clearing away the difficulty, notwithstanding the spirit of wisdom and justice, and the great knowledge and authority with which it is written. Indeed, so far as it widens the legal view of irresponsibility on the ground of insanity, it certainly will not tend to lighten the burthen of responsibility hitherto borne by our Courts in these arduous inquiries. For, so long as our judges are content to lay down the law in the simple and concise formula that a man who intentionally commits homicide, knowing the nature and quality of the act, and that it is wrong, is punishable as a murderer, the law is not difficult to be enunciated; it is only difficult to apply, because it does not conform to the facts of human existence. But if the control of conduct, as influenced by disease, be admitted, as eventually it must be, into the consideration of guilt or innocence, an element of even greater intricacy than that of knowledge of the nature and quality of the act, or that it was wrong, will be added to the law of insanity, and although justice will be rendered more secure, its determination will not be facilitated.

There would seem to be three sufficiently distinct modes of considering the irresponsibility of homicides, in whose defence the plea of insanity is advanced. First, there is the old customary manner of stating the law, which requires the defence to prove inability (arising from insanity) to know the nature and quality of the act, or that it was wrong; and there is some doubt as to whether the word "wrong" means legally wrong only, or that it may mean morally wrong. Secondly, there is the proposed modification of the law, inability to control conduct caused by disease; and, Thirdly, there is what may be called the physiological, in contradistinction to the two first, which may be considered the legal modes of considering this question. The physiological mode we should say is to add to the considerations as capable of determining the question at issue, the state of the man's mind with regard to the particular act, as to whether he knew its nature and quality, or whether it was wrong legally or morally, or whether he was in fact and deed capable of resisting the desire to commit it, the consideration whether or not the accused was or was not at the time of the act suffering from disease affecting his mental functions so as to render him generally incapable of reasoning, feeling and acting like other men. And it is not presumptuous to affirm that this is the only mode in which the mental results of disease can be considered exhaustively. To a certain extent it includes both of the legal modes, but it is by no means merely commensurate with them. For while no insane man really knows the nature and quality of any of his actions, in the full sense of the knowledge of a rational man, and no insane man recognizes as a rational man does the full extent of the wrong he commits by a criminal act; at the same time, a large proportion of the insane do know to a great extent what they are doing and whether it is wrong, and also they can very frequently resist very strong temptations, whether they be of a natural or morbid kind. There is no mental function which is not liable to be affected by an attack of insanity, neither is there any mental function which is usually quite suspended or destroyed by such an attack of insanity as is likely to leave a man capable of committing an offence and likely to be punished for it. Moreover, there are mental functions affected, or disabling mental states produced by disease which can scarcely be connoted by any meaning of the term "knowledge." It would seem, therefore, that there is no mental test which lawyers or legislators can adopt which is not liable to fail if it be made narrow, definite and absolute. Such tests, for instance, as does not know, cannot help it, cannot control his conduct, when, in fact, he does know to a great extent, can help it, and can control his conduct, if the inducement to do so be sufficiently strong and urgent.

The author declares, and there can be no doubt that he is right, that "the question, What are the mental elements of responsibility? is and must be a legal question. It cannot be anything else, for the meaning of responsibility is liability to punishment; and if criminal law does not determine who are to be punished under given circumstances, it determines nothing."

But the question before a Criminal Court, is not what are the elements of responsibility, but whether these elements do or do not exist in the individual accused of an offence. And when it is affirmed that these elements are absent, in consequence of bodily disease affecting the prisoner's mind, the question becomes not a legal but a medical one; and this the author to a certain extent acknowledges. He says, "I think it is the province of the medical men to state for the information of the Court such facts as experience has taught them bearing upon the question whether any given form of madness affects, and in what manner, and to what extent it affects, either of these elements of responsibility, and I see no reason why, under the law as it stands, this division of labour should not be fully carried out."

But this division of labour would render medical evidence general and impersonal, and of less real value in determining innocence or guilt than the opinion of any common man, a ploughman or a sailor, who had known the accused and could testify as to what he thought of him. The author must therefore have intended, in the division of labour which he approves, to indicate the evidence which a physician may be permitted to give who has made no personal observation of the individual accused. This kind of evidence is now very properly restricted in criminal trials to the generalities with which it deals, which have a certain value, but which are not comparable to the evidence of detailed personal observations made by competent medical men, which ought always to be available in so grave an inquiry as the responsibility of a murderer defended on the plea of insanity.

We take it, therefore, that the division of labour approved of by the author relates to the division of labour in the formation of opinion, more or less authoritative, in generalities. Firstly, there is the almost absolutely authoritative opinion of the judge as to the mental elements of responsibility; and, secondly, the opinion of the medical authority, as to whether a certain form of madness does or does not include those elements. For instance, the judge, with the authority of his high office, gives the jury what may be called the order that they must only consider as the mental elements of responsibility the knowledge of the nature and quality of the act and that it was wrong. And the physician who has not examined or personally known the accused, may be asked whether a state of mental depression without insane delusions is inconsistent with those mental elements, and it would not be permissible for the physician in such a case to state his opinion that there were other elements of responsibility, than those laid down by the judge.

Unfortunately the opinions of medical men on responsibility vary widely, according to their character, education and experience, but the law of the matter, that is to say the mental elements of responsibility, ought to be absolute and definite. This, however, would seem to be by no means the case, and the uncertainty seems to be fully admitted by the author, who says:—

"I believe that by the *existing* law of England those elements (*i.e.* the mental elements of responsibility) so far as madness is concerned, are knowledge that an act is wrong and power to abstain from doing it. I think it is the province of the judges to declare and explain this to the jury." "If I am wrong in thinking that the power to abstain from a given act is an element of responsibility for it, the duty of the judge is to tell the jury that such is the law, and to exclude from the consideration of the jury as being irrelevant, all evidence leading to show that the accused person was deprived by disease of control over his actions."

It would appear from this that Sir James Stephen, a judge of the High Court of Justice, is not certain as to what is the law of England in this important matter, and those who have studied his fair-minded and candid pages will be the less surprised when they have observed other judges in recent trials laying down the law of England as he would not have done. On a quite recent occasion, for instance, Mr. Justice Day, in the trial of William Gouldstone, on the 14th September last, for the murder of his five children at Walthamstow, is reported in the *Times* to have charged the jury thus: "He told them as a matter of law, that if the prisoner, when he killed the children, knew the nature and the quality of the act he was committing, and knew that he was doing wrong, then he was guilty of wilful murder. The nature and quality of the act meant that the man knew what it was that he was doing, that is to say that he knew he was killing a fellow-creature. He repeated that if a man killed a fellow-creature, knowing at the time he was doing wrong, then he was guilty of wilful murder. The only question therefore for them to determine was whether the prisoner knew the nature and quality of the act he was committing, and whether he knew it was wrong." In this enunciation of the law on this most solemn occasion there was nothing said of that mental element of responsibility which consists in the power to abstain from a given act, and therefore either the "existing law of England" has been too widely stated in the author's pages, or it was most inadequately stated in Mr. Justice Day's charge.

That Mr. Justice Day's law was held by the judges generally to be the existing law of England up to a comparatively recent date cannot be doubted, for Lord Chief Justice Cockburn, in his Memorandum to the Select Committee on Sir James Stephen's Homicide Bill, in 1874, remarks: "As the law as expounded by the judges in the House of Lords now stands, it is only when mental disease produces incapacity to distinguish between right and wrong that immunity from the penal consequences of crime is admitted. The present Bill introduces a new element, the absence of the power of self-control. I concur most cordially in the proposed alteration of the law."

The law, however, has not been altered by statute, and it is an interesting and important question in what manner and at what time it has been or may be altered by the individual or collective action of the judges. The alteration will certainly meet with the concurrence and approval of the medical profession.

It is quite a novelty to find a work of this nature, scope, and authorship, founded and argued upon a large appreciation of medical opinion; although with regard to such opinion and authority the author is evidently, and perhaps necessarily, somewhat in the same position in which he describes medical men to be in with regard to the law, when he says:

"I must say that the provisions of the existing law have, as it seems to me, been generally, though perhaps not unnaturally, misunderstood by medical men, who cannot be expected either to appreciate the different degrees of authority to be ascribed to different judicial declarations of the law, or to understand the rules for their interpretation, or to recognize the limitations under which they are made, or to appreciate the fact that when made they cannot be altered at will. Moreover, to read judicial decisions correctly is an art in itself, to be acquired only by long professional practice, nor can any one even begin to do so before he has familiarised himself with several rules well known to lawyers, but, in my experience, altogether unknown to medical men."

Although this may not be quite true in its *vice versâ* application to the interpretation by lawyers of that medical science which we are so often blamed for rendering needlessly recondite and technical, still it is so far true that the balance of medical opinion can scarcely be fairly held by any hand which has not been trained in the gymnasia of physiological study. And this is peculiarly the case in a department of medical science so new and developing, and yet so incomplete, as that of mental pathology. But while the pages of our author afford ample evidence that the legal mind is capable of grappling with the most intricate and difficult medical questions, so far as his professional duties require them to be understood, he would, we are sure, admit that medical men who gave their minds to it, might attain to the power of correctly reading judicial decisions. An example of the author's professional view of insanity as distinguished from the physiological view, is seen in the tendency which he nowhere exactly expresses, but which makes itself felt throughout his pages, to consider insanity as one condition the development of which is explicable according to Griesinger, whom he largely quotes, on the Guislain theory of phrenalgia; and another example in his acceptance of the theory of moral insanity. Our limits do not permit more than the expression of what we believe to be the true physiological view, that insanity is but a symptom of a great number of bodily diseases, and that the theory of moral insanity promulgated on such insufficient grounds by Pritchard and Ray, is to be relegated to the long list of exploded errors which occur in the gradual development of a difficult science. But considering the yet existing imperfection of this science, and the bias which the legal mind almost necessarily has, to consider exclusively what are called the mental elements, the ampleness of the conclusion arrived at by the author is, it must be admitted, far in advance of all former legal authorities. This conclusion is:

"As to the verdict of not guilty on the ground of insanity, the foregoing observations show in what cases it might in my opinion be returned; that is to say, in those cases in which it is proved that the power of self-control, in respect of the particular act, is so much weakened that it may be regarded as practically destroyed, either by general weakening of the mental powers, or by morbid excitements, or by delusions which throw the whole mind into disorder, or which are evidence that it has been thrown into disorder by diseases of which they are symptoms, or by impulses which really are irresistible and not merely unresisted."

This summing up of the mental elements of irresponsibility appears to us admirable, although we can scarcely admit that even yet it is perfect. Perhaps, for instance, the addition of morbid defects to morbid excitements would widen its scope and strengthen its significance. It is however the most comprehensive condensation of what ought to be the law which we have ever met with by any authority, legal or medical, and it is especially to be noted that its whole structure is based upon the foundation of the loss of self-control, thus differing in an essential respect from the author's former attempt to formulate a reasonable and satisfactory dictum.

For in Sir James Stephen's Homicide Bill, which was the subject of the Select Committee in 1874, he proposed to codify the law in the following manner:

"24. Homicide is not criminal if the person by whom it is committed is at the time when he commits it prevented by any disease affecting his mind. *A.* from knowing the nature of the act done by him. *B.* from knowing that it is forbidden by law. *C.* from knowing that it is morally wrong; or, *D.* from controlling his own conduct. But homicide is criminal, although the mind of the person committing it is affected by disease, if such disease does not in fact produce some of the effects aforesaid in reference to the act by which death is caused, or if the inability to control his conduct is not produced exclusively by such disease. If a person is proved to have been labouring under any insane delusion at the time when he committed homicide, it shall be presumed, unless the contrary appears, or is proved, that he did not possess the degree of knowledge, or self-control hereinbefore specified."

In our Lumleian Lectures of 1878, on Insanity in its Legal Relations, we recognized the above "as the only earnest and serious attempt which has been made to amend this stereotyped fragment of the common law," but we proceeded to criticise it as follows:

"It is evident that the pith and substance of this scheme of law is comprised in its fourth section, which, like Aaron's rod, turned into a serpent, swallows up all the others. If we complete each sentence my meaning will be apparent. The full expression of exemption ought clearly to be as follows. Homicide is not criminal if the person by whom it is committed is, at the time when he commits it, prevented by any disease affecting his mind from knowing the nature of the act committed by him and therefore from controlling his own conduct. Between the not knowing and the doing there is a great gulf fixed, which can only be bridged by words conveying the idea of motive; the words used 'prevention of control' being most convenient.

"From this consideration it appears to me demonstrable, that clause D of the proposed code virtually includes all the others, and is sufficient in itself. 'Homicide is not criminal if the person by whom it is committed is, at the time when he commits it, prevented by any disease affecting his mind from controlling his own conduct.' This could scarcely need any further development in terms, if it were only wisely acted upon in the full spirit of Lord Justice Blackburn's dictum, that, in every individual case, you must look at the circumstances, and do the best you can to say whether it was the disease of the mind, which was the cause of the crime, or the party's criminal will."

That this loss of self-control is not merely to be considered as an addition to the anciently recognised elements, but that it is the one essential though secondary element of irresponsibility, we pointed out in another of our Lumleian Lectures, as follows:

"There is one attribute of conduct produced by disease which is sufficiently simple to be intelligible by men of plain common sense, sufficiently constant not to cheat us with false hopes of guidance, and directly applicable to the end in view, and this is the uncontrollable character of conduct resulting from disease of the brain. Surely it must be that this quality of conduct resulting from disease has always been at the root of the law, although even up to the present time it has never been expressed in the law; for if this quality be abstracted, what reason would remain for excusing a man from punishment, on account of ignorance or unreason, even if he be insane? If a madman could be made to control his conduct like other men, we can scarcely believe that any tenderness for his misfortune would be able to overrule the obvious expediency of compelling him to do so; while in his deprivation of self-control, and his insubjection to imposed control, we have a test of his madness and a practical reason for his irresponsibility."

We insist greatly upon the importance of assigning to loss of self-control from mental disease its proper place and function as the secondary but immediate and exciting cause of the homicidal act, while ignorance and delusion have assigned to them their proper place and function as remote causes, because it appears to us that to admit loss of self-control, without reference to other mental states intermediate between the diseased organism and the action as a ground for irresponsibility, would be inconsistent with psychological truth and dangerous to the administration of justice, by sanctioning the widest theory of insanity of the emotions without any trace of intellectual aberration or defect. It is therefore with great satisfaction that we note the decided change which has taken place in this respect in the author's proposed enunciation of the law since he drew his Homicide Bill some ten years ago. The principle being admitted, that loss of self-control caused by disease primarily affecting the intellectual functions is the one constant element of the irresponsibility of the insane, the most convenient dictum in which it can be expressed will no doubt be eventually eliminated, if it has not already been attained by the author in the passage last quoted from his most recent work. Perhaps it may be that the best formula of words, or even the truest ideas, have not yet been quite reached. Perhaps other modes of mental diseases in addition to weakness, excitement, and delusion, may be recognised;

and very likely the uncomplicated existence of "impulses which really are irresistible and not merely unresisted" will be found an idea too vague and uncertain to be admitted into the administration of the criminal law.

We have to observe another important improvement in the author's present suggestion of the law as it ought to be, in his omission of all circumstances which render "a homicide criminal, although the mind of the person committing it is affected by disease." Some of these, as stated in the Homicide Bill, are tautological, but the provision which affirms that homicide by a person affected by mental decease is still criminal, "if the inability to control his conduct is not produced *exclusively* by such disease," appears to us quite unworkable. What action of any lunatic can ever be predicated as produced exclusively by disease? Clearly none. The knowledge which a man has acquired before he has become insane goes largely to the direction of his conduct, for, as far as knowledge is concerned, the maddest action is more the result of sanity than of insanity, if it be possible, even theoretically, to discriminate different portions of a man's mind at the same moment into the two states. And, in practice, how would it be possible to say of an insane drunkard that so much of his conduct was due to madness, and so much to drunkenness? Take as an example the lunatic who shot a provision dealer at Holloway, and was found guilty, we believe, because it was proved that he was drunk at the time. It was, however, pointed out to the Home Secretary that this man had attempted to gain access to his victim, for the purpose of homicide, three or four hours before he actually did commit it, and when he was quite sober, though at both periods he was under the influence of insane delusion; and the man was very properly respited. Such cases are not rare, nor the still more difficult ones in which an obviously insane person is influenced to a criminal act by a natural but malicious motive; a motive, however, which would not have led to its actual result but for the mental disease; and it must be impossible for human intelligence in such a case justly to apportion the blame and the punishment. We are glad, therefore, to find that the author in his most recent dictum omits this impracticable condition of irresponsibility. Another somewhat similar question discussed by the author, although wisely omitted from his dictum as to what the law ought to be, is to what extent any lunatic ought to be held responsible for his madness, and whether the disease which causes the mental elements of irresponsibility ought to be defined as disease not occasioned by his own default. He states his grounds for this suggested alteration of the law as follows:

"In reading medical works, the connection between insanity and every kind of repulsive vice is made so clear that it seems more natural to ask whether in many cases insanity is not rather a crime in itself than an excuse for the crimes which it causes. A man cannot help an accidental blow on the head; but he can avoid habitual indulgence in disgusting vices, and these are a commoner cause of madness than accidents. He cannot avoid the misfortune of being descended from insane or diseased parents; but even if he has that misfortune, he ought to be aware of it, and to take proper precautions against the effects which it may be expected to produce. We do not recognise the grossest ignorance, the most wretched education, the most constant involuntary association with criminals, as an excuse for crime; though in many cases—I think in a smaller proportion of cases than is commonly supposed—they explain the fact that crimes are committed. This should lead to strictness in admitting insanity as being in doubtful cases any excuse at all for crime, or any reason for mitigating the punishment due for it. It is upon this ground that I think that the general rule that a person should not be liable to be punished for any act done when he is deprived by disease of the power of controlling his conduct should be qualified by the words "unless the absence of the power of control has been caused by his own default."

This proposal is simply impracticable, for with the exception of the few cases of traumatic insanity referred to, are not most cases of insanity more or less caused by the patient's own default, in the author's sense of default? that is to say, that the person liable to its development ought to know his danger and to take proper precautions to avert it. When it is remembered that the disgusting vices which are a common cause of insanity are themselves very frequently the results of a lower degree or less developed form of mental disease; and when it is remembered that hereditary insanity is oftentimes a gradual development of obscure conditions from infancy upwards, it may safely be affirmed that it is beyond the reach of the highest human wisdom, and certainly far away beyond the rough and ready methods of our courts of criminal law, to distinguish how much of a man's madness is the result of his own default. And if it were the result of his own default, ought he not in justice to be punished for his madness which by the proposition he ought to have foreseen and avoided, and not for his criminal offence, which, theoretically, has been caused by the default of others in not having recognised the man's madness and taken due precautions against its results? And is not madness itself a sufficient punishment for the offence of neglecting one's health?

There are several other questions mooted and argued with vigorous common-sense, cleansed from all sentimentality, which seem to invite a reply, in which we should greatly like to see what could be said from the medical point of view, if our limits would permit. But we would conclude by strongly recommending Sir James Stephen's pages, not to the perusal merely, but to the diligent study of any medical man who may wish to obtain some mastery of one of the most arduous duties to which the incidents of his professional life render him liable. They will not, it is true, give him much knowledge of mental disease, but they will enable him to arrange such knowledge as he may otherwise have acquired, in such manner as to make it available for the most grave and special purpose of giving evidence with understanding. They are bound also to have an important effect upon future legislation, or, failing that, upon the unwritten law of the land. They cannot be the last words on the subject, for they are too suggestive to be accepted as a settlement of many of the questions which they argue, but they very evidently constitute a new starting-point for anything of importance which can be said on the subject in the future.

JOHN CHARLES BUCKNILL.

Lehrbuch der Gehirnkrankheiten für Aerzte und Studirende.—Von Dr. C. WERNICKE. 3 vols. Fischer; Kassel, 1881–1883.

THE work before us is an exceedingly important one, and demands more than usual consideration at the hands of a critic. It aims at fulfilling the double function of an extended treatise on the diseases of the brain for the use of physicians, and a handbook for students. This two-fold object is endeavoured to be attained by the use of large and small type. The leading features of the anatomy, and the fundamental principles of the physiology of the brain, as well as the systematic description of the clinical phenomena, are printed in large type; whilst the details of the anatomy and the debateable questions of the physiology of the brain, as well as the reports of all the illustrative clinical cases, are printed in small type.

The work, so far as yet published, consists of two volumes of somewhat unequal size. The first volume is divided into two parts: in the first part, which extends over 272 pages, a full and elaborate account is given of the anatomy and physiology of the brain, and in the second part, which occupies the remaining 100 pages of the volume, the individual symptoms of cerebral disease are described and explained. The second volume, which consists of 251 pages, is occupied with the consideration of the general pathology of focal diseases, but the only diseases of this class which the author has discussed up to the present are hemorrhage and softening. It will thus be seen that this work is a mere fragment of what we may expect it to attain. The wide subject of intracranial tumours has yet to be dealt with, and we may rest assured that the author will not decline the consideration of the diffused inflammations of the brain and of its membranes, or such affections as shock, concussion, neurasthenia cerebralis, epilepsy, and hysteria.

The first part of the first volume, which treats of the anatomy and physiology of the brain, is by far the most important section of the work. This part occupies rather less than one half, and considerably more than one third of the whole book, and is illustrated by 96 woodcuts. Of the woodcuts, 21 are purely diagrammatic, and illustrate the text which is printed in large type, and which is intended for the use of students. These diagrams are admirably conceived, and they render the course of the various communicating fibres and conducting paths as clear as the complicated nature of the subject will permit. Of the remaining figures a few are borrowed from Flechsig, Charcot, and Munk, but by far the greater number are original, and are drawn from nature. Everyone accustomed to examine horizontal sections of the hemispheres, the crura, pons, and medulla oblongata will at once recognise the extreme fidelity of these figures; nothing can exceed their delicacy of execution, and they do equal credit to the artist, the engraver, and the printer. The lines are so delicate, and for the most part so well defined, that the figures look more like steel engravings than woodcuts. In a few of these figures, however, a somewhat bolder treatment would have been desirable, the representation of a section of the lower end of the medulla oblongata [Fig. 69] being, for instance, so indefinite that it is difficult to trace any structure whatever.

The account which Wernicke gives of the anatomy of the brain differs in some noteworthy respects from that of other recent authors, while his own original contributions to cerebral anatomy are by no means few or unimportant. In describing the surface of the frontal lobe the author, following the earlier anatomists, such as Leuret, numbers the convolutions of the upper and outer surface of the lobe from below upwards, instead of from above downwards as is done by Wagner and Ecker. If the reader fail to remember this, he will be startled to find it asserted that motor aphasia is caused by lesion of the posterior extremity of the *first* left frontal convolution.

The longitudinal or collateral system of fibres are usually said to consist of (1) arcuate fibres or fibræ propriæ, (2) fibres of the gyrus fornicatus, (3) the fasciculus uncinatus, and (4) the inferior longitudinal fasciculus.

In addition to these, Wernicke describes for the first time a large bundle of fibres which, from the course they pursue, he names the *perpendicular occipital fasciculus;* these fibres connect the inferior parietal lobule and the fusiform lobule of the temporo-sphenoidal lobe. Nothing beyond conjecture can as yet be said with regard to the functions of this fasciculus. The author asserts that neither the caudate nucleus nor the external division of the lenticular nucleus is directly connected with the cortex by radiating fibres. He believes that these masses of grey matter must be regarded as independent ganglia, like the grey matter of the cortex itself. The first and second divisions of the lenticular nucleus he regards as ganglia of interruption, which connect the caudate nucleus and the third division of the lenticular nucleus with the central grey tube.

The opinion of Broadbent, Charcot, Flechsig and others that the fibres of the pyramidal tract pass uninterruptedly through the internal capsule or crusta is confirmed by Wernicke. The most noteworthy contribution which he has made to the elucidation of the constitution of the internal capsule, is his statement that those fibres of the corpus callosum, which form the anterior wall of the anterior horn of the lateral ventricle, wind backwards along the external border of the caudate nucleus to mingle with the descending fibres of the anterior segment of the internal capsule. The fibres of the pyramidal tract are said by Wernicke to be everywhere interrupted in the pons by grey matter, but we do not share this opinion. An attentive examination of sections, especially from the embryo, and from cases of descending degeneration, will, in our opinion, show that, although the longitudinal bundles of the pons are often surrounded by grey matter, yet the fibres do not form connections with the ganglion cells of this substance.

The connection between the basal ganglia and the various tracts and ganglia of the crura cerebri are very carefully described, and illustrated, the diagram [Fig. XII.] which explains the connections between the different parts of the optic thalamus and the tegmentum being particularly instructive. The author has traced connecting fibres between the optic thalamus and the red nucleus of the tegmentum. But a case of descending degeneration secondary to a focus of softening in the pulvinar of the optic thalamus has recently been reported by Mendel,[1] which seems to show that these fibres pass along the inner margin of the red nucleus, and, then cross over to reach the superior peduncle of the cerebellum of the opposite side, where they could be followed as far as the corpus dentatum of the cerebellum. Connecting fibres between the optic thalamus and the fillet have been discovered by Wernicke. He also states that the innermost fasciculus of the fillet is derived from a bundle of fibres which is situated on the internal border of the crusta; this fasciculus does not reach the medulla oblongata, but terminates in the pon in grey matter.

The tract of fibres which lies close to the nucleus of origin of the third nerve, near the floor of the aqueduct of Sylvius, named the *posterior longitudinal fasciculus*, is said by Wernicke to be connected with the first division of the lenticular nucleus by means of the fibres which form the anterior portion of the collar of the crus. The posterior longitudinal fasciculus maintains throughout the whole length of the crus, pons, and medulla oblongata a close connection with the nuclei of origin and roots of the motor nerves, and it is continued downwards in the spinal cord as the inner division of the anterior root-zone. If this fasciculus is, as Wernicke asserts, connected with the lenticular nucleus, and not with the optic thalamus, as has hitherto been supposed, an additional argument is afforded in favour of the opinion that it is endowed with motor functions.

The fibres of the internal bundle of the optic tract are said by Wernicke to turn upwards in the external part of the crusta to reach the posterior portion of the internal capsule, and without forming any connections with either of the geniculate bodies or with the anterior tubercle of the corpora quadrigemina. Other noteworthy discoveries made by the author are that the fibres of the posterior bundle of the anterior commissure of the third ventricle terminate in the cortex of the temporo-sphenoidal lobe, and that the lenticular nucleus is connected by fibres with Luys' disc and with the locus niger.

These, then, are the chief contributions which Wernicke has made to the anatomy of the brain, and it will be seen that they are both numerous and important. No part of the anatomy of the brain, however trivial it may appear, is without its value to the physician who knows how to decipher its meaning, and the man who possesses the most accurate knowledge of the structure of the nervous system in its minutest details, is likely to be the best clinical observer, as well as the best interpreter of what he does observe. Dr. Wernicke has now placed within our reach the fullest and best account of the structure of the brain, for clinical purposes, which has hitherto appeared in any language, and for doing this, special workers in the field of neurology owe him a deep debt of gratitude, which they will be forward to acknowledge.

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[1] Mendel, "Secundäre Degeneration im Bindearm;" 'Neurologisches Centralblatt,' Bd. 1, 1882, p. 24.

The physiological part of the first section of the book, does not probably stand out so pre-eminently for its excellence as the anatomical part, but the account which the author gives of the experimental investigations of Fritsch and Hitzig, and of those of Munk, is good, and sufficiently full to form a ground-work for the interpretation of the clinical phenomena. The author also makes good use of the phenomena of the different forms of aphasia, which he divides into motor, conductive, sensory, and total aphasia, for the elucidation of the functions of the brain. A very useful account is likewise given of the course of the motor and sensory paths, which connect the cortex of the brain with the periphery. He adopts the views of Flechsig and Charcot in his description of the course of the motor conducting paths, while his discussion of the different routes which the sensory conducting tracts may possibly pursue, in passing from the spinal cord to the internal capsule, will be found to be exceedingly instructive.

Want of space forbids us to enter upon a detailed examination of the clinical part of the work. We have already seen that only a very small proportion of the diseases of the brain have as yet been described by the author, and we must give it as our opinion that the clinical portion, although full and good, hardly equals in excellence the anatomical and physiological section. It may be noticed in passing, that the author assents to the doctrine that epileptic convulsions are caused by irritative lesions of the cortex of the brain, and not by lesions of the medulla oblongata. It need scarcely be added that the theory of cortical discharge first enunciated by Hughlings-Jackson, and subsequently confirmed by the experiments of Ferrier, has for a long time been adopted by neurologists in this country.

We shall now point out what we conceive to be a few of the shortcomings of this work. This part of our task is undertaken in no carping spirit, and with no desire to detract from the great value of Dr. Wernicke's labours. We offer our remarks, indeed, with great diffidence, and with the full consciousness that the author may be, as so often happens, wholly in the right, and his critic wholly in the wrong. The first objection which we shall urge is, that there is an absence throughout the book of a fertile principle, like the law of evolution, and its converse the law of dissolution, which ought to run like a warp through the entire tissue of facts, giving strength to the texture and unity to the design of the work. This defect leads to a want of unity in the different parts, and the author has been unable to utilise his very excellent though brief account of the development of the brain towards the elucidation of the phenomena of disease. Embryology, which ought to afford the best clue for unravelling the mysteries of disease, is, in Wernicke's hands, almost unused and useless. The author, following Meynert, begins his description of the brain with the cortex, and thence passes downwards by successive horizontal sections to end with the description of the junction of the medulla oblongata with the spinal cord. Much may be said in favour of this mode of procedure, but we believe it to be greatly inferior to the reverse method, which begins with the upper end of the spinal cord and ends with the cortex. The method adopted by Wernicke begins with the most complicated part of the brain, and ends with the simplest, but it would surely be wise to begin, after nature's order, with the simple and to proceed gradually to unfold the complex. It is manifest that the simplest portion of the brain is to be found in the embryological tracts of the spinal cord at their junction with the medulla oblongata, and consequently the best method for unravelling the complex structure of the organ, would be to follow these tracts to their various terminations in the medulla oblongata, pons, and crus cerebri, and then to trace their connections with the higher ganglia, and the connections of those ganglia with still higher ganglia, until ultimately the cortex is reached.

It is to be regretted that Wernicke has limited himself almost entirely to horizontal sections, and the introduction of Pitres' vertical sections of the cerebral hemispheres into this work would, in our opinion, be a great improvement.

In the clinical part the author discusses in full the pathology of focal diseases, first when the lesion is a hemorrhage and second when it is a softening. According to this method of procedure a great deal of what is said with regard to what may be called the localising symptoms in the section on hemorrhage has to be repeated in the section on softening, and will have to be repeated a third time when the author comes to deal with the subject of intracranial tumours. We believe that it would not only be an economy of space; but that it would also conduce much to clearness of treatment, were the author to discuss at first the general symptoms of hemorrhage, softening, and tumours separately, and subsequently describe the special or localising symptoms of all these lesions in one section. Many of the symptoms of disease of the lenticular nucleus, for instance, will be the same whether the lesion be hemorrhage, softening, or tumour, and nothing but confusion can arise by describing these symptoms separately for each lesion. Probably the most serious blot on the whole work, is to be found in the fact that the description of the blood vessels of the brain, according to the researches of Heubner and Duret, is to be found as a kind of appendix to the second volume, and consequently after instead of before the consideration of the subjects of hemorrhage and softening. The absence of illustrative diagrams of the arteries of the brain is also a serious drawback.

We observe with pain that, although a somewhat grudging acknowledgment is made of the value of Ferrier's experimental determination of the cortical motor centres of the monkey, yet what we conceive to be his more important investigations of the sensory cortical centres is passed over with significant silence. It is possible that the experiments of Ferrier and Yeo on the occipital lobes may not have been published before Wernicke's work had passed through the press, but this cannot, of course, be said of Ferrier's earlier experiments for the determination of the sensory centres. We are aware of all that can be said against the validity of these experiments, but the facts still stand that Ferrier was the first physiologist who broke ground in this direction, and that the conclusions at which he arrived, from the admittedly imperfect data at his command, are marvellously accurate, even when regarded from the standpoint of our present more advanced knowledge. We know that there are several reasons why Ferrier's work should be less appreciated at Berlin than elsewhere, and all we can say is, that after giving full weight to these reasons, and after making a strenuous endeavour to place ourselves in the attitude of a Berlin physiologist with relation to the facts, we are totally unable to sympathise with the spirit which has dictated this want of generosity towards Ferrier and his work.

In bringing this review to a close, we may be allowed to express the opinion, that even after giving due consideration to every fault and shortcoming which the most captious critic can point out, the work before us must still be regarded as an extremely valuable one, and a noble contribution to neurology. These volumes are, indeed, well worthy of the great school of medicine from which they have emanated; they cannot fail to enhance the already high scientific reputation of their distinguished author, and we feel assured that for many years to come they will not be found far from the elbow of all who have to lecture upon, or make a special study of the diseases of the nervous system.

Since the foregoing remarks were written the third volume, or rather the second part of the second volume, of Dr. Wernicke's work has come to hand. It deals with the subjects of intracranial tumours, abscess of the brain, diffused multiple sclerosis of the brain (chronic polyencephalitis), the various forms of cerebral meningitis, idiopathic hydrocephalus internus, thrombosis and phlebitis of the cerebral sinuses, and progessive paralysis of the insane. This portion of the work extends over 300 pages, and the various subjects comprised in it are discussed in a very elaborate and satisfactory manner, each disease being fully illustrated by valuable records of clinical cases. So far as we can judge from a necessarily cursory examination, the new volume attains a higher level of excellence than has been reached in the clinical portion of the earlier volumes.

JAMES ROSS.

The Brain, considered Anatomically, Physiologically, and Philosophically.—By EMANUEL SWEDENBORG, Edited, Translated and Annotated by R. L. TAFEL, A.M., M.D., Ph.D. James Speirs, 36 Bloomsbury Street, London.

THIS is without doubt one of the most remarkable books we have seen, and perhaps to a great extent, on that very account, it is a very difficult task to criticise or review it. The distinguished author lives in history more as the founder of a form of religious thought than as a scientific man, and yet it is a fact that he was eminent as a mathematician, as an engineer, and as a mineralogist and geologist. Still it is a new experience to find him figuring as the writer of a work on the human Brain, and still more curious to discover that, although that work has lain by for nearly a century and a half, it is now not only not valueless, but it appears actually to have anticipated some of the most modern discoveries in the region of its subject-matter. Only the first volume of this remarkable work is before us, but we are bound to say that the prevailing feeling forced on our minds during its perusal has been one of surprise at the great power and profound philosophical insight of the writer, as point after point of his argument has been brought into view. No doubt, to Swedenborg, this work had mainly a theological interest. He was over fifty years of age when he wrote it, but it was about this time in his life that he directed his energies mainly into those lines from which arise his claims to live in the temple of fame, and the remaining thirty years of his life were employed in elaborating the views he had already formed. This fact undoubtedly makes it more difficult to gauge the value of the work, because for us it has only a scientific interest, and it is therefore necessary to unravel, and, so far as possible, to confine to its rigidly scientific bearing the phraseology, now metaphysical, now ontological, now theological, of which the author makes use. And indeed we may say that if it had not been that attention was arrested and interest enchained by finding so many anticipations of scientific discoveries by as much as 120 or 130 years, we should have been tempted to throw aside the book as beyond our province, if not hopelessly unintelligible. For it is certainly difficult to deal scientifically with such expressions as "essences of a superior and inferior kind," "the ideal of the universe which is below," "the supreme in every system of the soul," and such as "series determined into higher and lower spheres," with other like phrases which occur from time to time. In order, however, once for all, and so that repetition may be avoided—in order to make as it were the transition from Swedenborg's ontology to the scientific sphere, in which alone for the purposes of this review we are interested, it seems necessary in the briefest manner, to make some remarks which shall show at once the nexus and the separation between the two. Swedenborg assumes all through his writing the existence of the soul, which, according to him, has its seat in the minute parts (each as it were a smallest brain with its own vessel, its own grey substance, and its own fine membrane) of the cortical substance; and he always assumes, because he saw or felt it as an innermost conviction, that the soul was the representative in the body of God in the universe. This is, of course, a position, not unscientific indeed, but extra-scientific, that is to say outside the sphere of science. If it is a true position, if, that is, the Soul is a *vera causa*, or real existence, scientific inquiry will not indeed reveal results incompatible with its existence—this it cannot do if science keeps to the truth of phenomena, which is always her aim—but the phenomena with which alone science has to do will not and cannot reveal the presence of a *vera causa* like the soul, for this is not a phenomenon, but an existence *per se*. Language of an ontological character, therefore, used by a man who, like Swedenborg, was essentially an ontologist, or a man who strove to reach the domain of things in themselves, such language may fairly enough be studied by the man of science, that is by him who, for his present purpose, confines himself entirely to phenomena. All that the scientific man has to do—it is a thing simple enough in theory, though difficult in practice sometimes—is to make and repeat the mental reservation where ontological phrases are used, that the ontological assumptions are a method of explaining the phenomena, which may or may not be correct, but that the subject-matter of science is only the phenomena. This kind of correction however is not peculiar to the kind of consideration we have instanced—to views that is concerning the soul and God. It is fundamental to all science and to all philosophy, and in point of fact it is the consideration which in principle lies at the bottom of the division of the domain of philosophy into two schools, the intuitional and the experimental. An instance or two will explain our meaning, before we pass to the scientific side of Swedenborg's views concerning the brain. In causation, science knows only succession, or perhaps invariable succession; but invariability itself can be predicated only experimentally, that is to say that it always has been so—but theoretically there may be an exception any day, though no one expects that there will be. *Necessary* connection, science never can predicate, because necessity is not in phenomena, but only succession, invariable within the limits of experience. The same considerations apply to the *power* assumed to lie behind phenomena of causation as Hume's billiard balls proved to all the world in Swedenborg's own day. The scientific reader who is at all acquainted with the history of philosophy will at once recall many other instances of terms used freely by science, which are calculated to raise similar questions. Matter, Force, Energy, are some of them, as well as those we have mentioned, and it is curious to observe that though, properly speaking, these are philosophical terms, it is Science which has been responsible for the very much wider currency which of late years they have obtained. Of course where Science does make use of them it ought fairly to be only as general names and for the purpose of avoiding unnecessary repetition, but it is full of instruction to analyse the very various uses that scientific men make of such terms and to trace out the large and even appalling amount of very loose thinking which they frequently cover. But (and this is most important) for the very reason that *veræ causæ* like God and the Soul can never appear as phenomena, for this very reason science can say nothing about them—least of all can she say they do not exist. If they do exist, she cannot see them, because her vision does not extend so far—they are out of her range.

The questions relating to their existence are questions not for her at all, but for ontology, or for philosophy; so true is it that the metaphysical questions are after and also beyond the physical, the phenomenal, the scientific. We do not say what weight should be attached to the fact that terms involving reference to ontological considerations are continually arising out of science. Nor do we attempt to raise any ontological discussion on them. Our present purpose is only to show what we therefore repeat, that such a *vera causa* as the soul, assuming its existence, can never appear in phenomena. But the language of him who assumes a *vera causa*, because by the eye of reasoning he sees it, will be intelligible to the scientific man who refuses to transcend the phenomena, provided he always keeps in mind the difference that exists between phenomena and modes of accounting for them by the assumption of independent existing *veræ causæ*. We may add that the learned editor of Swedenborg appears, though without explicitly stating it, to have kept this great distinction continually in view, there being few instances indeed in which it has been forgotten. On p. 740, however, in the course of his very elaborate note on the Chymical Laboratory of the Brain, he uses "animal spirit," viz. the fluid secreted by the brain as synonymous with "nervous force," placed indeed within inverted commas, a phrase suggestive rather of metaphysical than of physical considerations. Again, when on p. 660, Dr. Tafel says, "The mind by its influx into the brain is the cause of the motion of that organ," does he use the word *mind* in a scientific sense or a philosophical?

The following are some of the views held by Swedenborg on the Brain and its functions, and the statement of them will show both how frequently he anticipated modern discoveries, and also how many of his opinions are still more or less peculiar to him and his followers. If time and space permitted, it would be very instructive to inquire in how many of these views Swedenborg was correct, or how far at least the evidence appears to justify the conclusions to which he came. Some of this evidence will be referred to in this review, but it will be impossible to take up all the points, interesting and important though some of them undoubtedly are:—

1. Swedenborg taught that the motion of the brain is coincident with respiration, not with pulsation. The origination of this view is now universally (that is so far as it is held, for many physiologists of the present time do not seem to know of it)

attributed to J. Daniel Schlichting, a physician of Amsterdam, who wrote in 1750; but Swedenborg wrote between 1741 and 1744. We propose to devote some space to the discussion of this view of Swedenborg, because it is fundamental to his opinions, and because the most elaborate and learned note of Dr. Tafel enables us to do this in a most complete way. We will at present therefore only say that the complete statement of Swedenborg's position on the subject is that the dura mater undoubtedly pulsates with the movement of the heart, but that the underlying brain as undoubtedly moves synchronously with the respiration and not with the pulsation of the heart.

2. Swedenborg held that the respiratory motion of the brain and lungs extends to the extremities of the body. This discovery is generally attributed to Dr. Piégu, who wrote in 1846.

3. Although Swedenborg taught that the movement of the brain was synchronous with that of the respiration, he also held that it is independent of it. This point will be discussed in our general review.

4. Any student of Swedenborg will see that the term "animal spirits," so frequently used by old anatomists and old philosophical writers, is an expression not used only of a metaphysical idea analogous to *soul*, but of a veritable fluid, having an actual existence, and composed, according to Swedenborg in a very definite way, and fulfilling a very definite function or purpose in the economy. We confess to having had our conceptions much cleared in this particular by a perusal of the book.

5. Swedenborg taught that the anterior part of the cerebrum is the seat of the intellect. This is precisely the view adopted by modern science.

6. It is worthy of mention that Swedenborg's division of the brain is not quite that which obtains at the present time. His highest lobe is bounded by the marginal convolution of the quadrate lobule, his middle lobe by the lower part of the marginal convolution, and the gyrus fornicatus; and his third lobe is identical with the temporo-sphenoidal lobe. Keeping this in mind, we find him making this statement. The muscles "and actions which are in the ultimate parts of the body, and thus in the soles of the feet, depend more immediately upon the highest portions of the cerebrum; upon the middle lobe the muscles of the abdomen and thorax; and upon the third lobe those which belong to the face and head; for they seem to correspond to one another in an inverse ratio." The general correspondence of these statements with the positions of the most modern science cannot fail to strike the reader.

7. Swedenborg says, "The corpora striata are vicarious cerebra, and they succeed in the place of the cerebrum whenever it is deprived of its power of acting." And again: "The corpora striata initiate movements which at first originated with the cerebrum, and were voluntary; for it is a well-known fact that voluntary acts by daily habit become spontaneous, or that habit is like second nature." This language again is strikingly like that of modern science.

8. So also Swedenborg showed the connection of the corpora

quadrigemina with the sense of sight; and also what we may shortly call the commissural arrangement of the corpus callosum.

9. Key and Retzius have shown (1875–6) that the choroid plexuses of the lateral ventricles are made up of two leaves, of which the upper comes from the fimbria of the fornix and the lower one partly. Strange to say, Swedenborg said very much the same thing 135 years before.

10. Swedenborg says that in order that the brain may be relieved of the liquid which is perpetually expressed from its members and arterial vessels, the above liquid passes into the cavities of the nose through the openings in the cribriform plate of the ethmoid bone. This position also, though it is not accepted by medical men or by physiologists, appears to have been corroborated by experiments made by Key and Retzius.

On this we will only say, since space does not allow of our discussing the question fully, that there are cases coming from time to time under the notice of the medical man which appear to corroborate Swedenborg's statement. Patients frequently say (we have met such) that they are subject to a form of attack accompanied by pain in the frontal region, generally brought on by over-mental work and simulating a cold in the head. Such attacks pass off, and relief is at once experienced on the appearance of a clear discharge from the nostrils. Such persons have assured us that they were also subject to attacks of nasal catarrh, and that they could perfectly distinguish between the two, but that the attacks now spoken of were quite different, and that in their opinion the fluid comes from the inside of the head and not from the nasal mucous membrane.

11. Swedenborg held that the active parts of the brain are the cortical substance, and that "the brain is made up of as many similar forms and natures as it has discrete cortical parts." And further that "the cortical substance is so connected with the minute vessels of the brain, that you would believe that the cortical glands derive their origin from the vessels." Swedenborg did not say he had seen this structure, but, reasoning from analogy, he felt certain of his position that "the cortical glands hang down," and sprout forth from the sides and coatings of the producing and generating arteries, scarcely otherwise than as grapes and berries are wont to hang down," &c. Dr. Bevan Lewis, writing in 1877, says from his microscopic investigations, that a minute blood-vessel invariably runs in close contact with all the large nerve-cells; and that the peri-cellular sacs are laterally disposed along the sides of the smaller capillaries, in no case occupying a terminal position. This is a remarkable corroboration of the truth of Swedenborg's statements.

Lastly. Swedenborg's method of enquiry and the conclusions at which he arrived appeared to him to demand that some foramen or foramina must exist at the base of the fourth ventricle, through which the cerebro-spinal liquid is discharged from the ventricle; and they also seemed to him to require the presence of a channel from the fourth ventricle into the spinal cord. Since Swedenborg's time a foramen in the former position has been discovered, called the foramen of Magendie, and also in the latter, where it is known as the central canal of the spinal cord.

As regards propositions 1, 2, and 3, on the Respiratory Motion of the Cerebrum, no mention whatever is made of this by such modern physiologists as Todd and Bowman, Carpenter, Michael Foster, Draper, Flint, Hermann or Ferrier. So that, speaking generally, we may say the view is not held by modern science. And yet Realdo Colombo (1559), Bartholin (1673), Wepfer (1681), Vieusseus (1685), Baglivi (1701), Ridley (1703), Bellini (1714), Pacchioni (1721), and Fantoni (1738), all of them literary predecessors of Swedenborg, had held and taught this view.

The question thus arises: how did a view held so widely come to be no longer accepted? From the learned note of Dr. Tafel we learn the answer to this question. It was Pelletan, professor of physics in Paris, 1829, who, basing himself on the impossibility of the walls of the cranium lending themselves to the least extension, also on the state of repleteness in which the cranial cavity is at all times, and on the incompressibility of the liquids and the cerebral substance, laid down, *à priori*, this proposition, that *in the normal state with the adult these movements are an impossibility.* In short, Pelletan held and Bourgougnon (1839) thought he had proved experimentally that in the air-tight cranium, filled with incompressible fluids, and with brain substance which behaved as such, all motion was impossible. The so-called proof of the proposition was this. Bourgougnon trephined a hole in the cranium, and then screwed a ferrule to which a brass tube had been inserted, into the hole. In the interior of the tube there was a little metallic plate resting on the brain, which communicated the motion of the brain to a little lever with an elbow, answering for an indicator. This motion was seen to agree nicely with the beating of the heart, so long as the tube containing the lever was open. But as soon as it was filled with water (previously boiled, in order to exclude its air-particles) beyond a little tap fixed so as to intercept all communication with the atmosphere, on the tap being turned off, the movements of the little lever at once stopped. This gave rise to the conclusion that the motion of the brain exists only as long as there is an artificial communication with the atmosphere. Richet, writing in 1860, said he had repeated Bourgougnon's experiment, and found it correct; but argued that the inductions drawn from it were false, and that the experiment did not by any means prove what it was assumed to prove. He pointed to the condition of the dura mater in the vertebral cavity, and to the existence in its cervical portion of various venous plexuses which are backed by a compressible layer of a fatty liquid, in order to prove the capacity of the vertebral tube of expanding. But an experiment devised by Mosso (1877) seems to settle the question, and in the words of Dr. Vaillard, to have established the motion of the brain in the ossified cranium. He says, "Put your hand into a jar (through a lid made air-tight) furnished with two mouth-pieces. One of these mouth-pieces is closed with a very elastic membrane; the other supports a tube with a tap, the tube being filled with water beyond the tap. It is easy to see that as long as the communication with the external air is kept up, the oscillations of the water consequent upon the increase and decrease in the volume of the hand are considerable in the tube where the tap is kept open, while there are scarcely any vibrations in the mouth-piece covered with an elastic membrane. Why is this? Evidently because the changes in the level are produced at that point where there is the least resistance, and because there is nothing to oppose itself to the manifestation of these changes in the tube which is in communication with the open air. On the other hand, the elasticity of the membrane of india-rubber covering the other opening, however weak it may seem, nevertheless opposes a serious obstacle to the movements of the column of water, all of which, in this case, are conveyed towards the open tube. This corresponds to the first part of Bourgougon's experiment, to that part where the movements of the brain are conspicuous. Let us now close the tap of the tube in which the oscillations had been so manifest. All motion now disappears, and it cannot be otherwise, since the column of water is not compressible. But has your hand on that account ceased to change in volume, and to swell each time when there is an afflux of blood? All you have to do is to examine the elastic membrane, which has hitherto remained motionless, and which now begins to heave and to subside by turns, in order to convince yourself of the fact that the oscillations still continue, and again manifest themselves where they meet with the feeblest resistance. The case is the same in the second part of Bourgougnon's experiment; if the lever which rests on the brain ceases to indicate any motion after the tap is closed, the reason is that the movements are conveyed towards those parts of the vertebral canal which are compressed."

The motion of the brain being thus established as a fact, the next consideration is of what sort is that motion. Up to the time of Swedenborg, although, as we have seen, there was no doubt about the motion (which was doubted only after his time) it was universally (?) held that that motion was coincident with pulsation. Swedenborg's views led him to the conclusion that it must be coincident with the respiration. He knew well enough that there was a motion of the brain coincident with pulsation also, but that motion he held was due to the dura mater, or rather to the outer layer of that membrane. The movement of the inner layer of the dura mater, on the other hand, corresponded with the motion proper of the brain and was coincident with the respiration. Now, in these particulars, it is very remarkable that later discovery has entirely justified Swedenborg's views. Some time ago the present writer abstracted for 'BRAIN' Professor Mosso's work on the motion of the brain, referring at length to the tracings obtained from that motion by the graphic method of Professor Marey. There is therefore the less reason to do here more than refer to his work, but it may be said that the labours of all the inquirers, by means of Professor Marey's graphic method—a method about which there can be no mistake, since it is really self-registering, coincide in showing changes in the volume of the brain coincident with the respiration. There is no doubt about the pulsatile movement of the dura mater, but coincident with this there is also observed a heave and fall of the curve corresponding with the respiration. Dr. Tafel reproduces in his note tracings from Salathè (1876), François-Franck (1877), and Mosso (1881), which show this double motion beyond dispute. Nor is there any more inherent difficulty in understanding how such a double movement may take place, than in understanding, what every one knows, that the heavenly bodies have a movement round a centre such as the sun, at the same time that they revolve on their own axis.

Leaving this point, then, we pass on to say that Swedenborg held that the respiratory motion of the brain, although coincident with it, was not due to the respiration, but was inherent in the brain itself. Here also modern science confirms Swedenborg's position, for Professor Mosso shows that a change in the pulse of the brain (observed in the dura mater) does not depend by any means upon a change in the movements of respiration. "Thus" he says, "while I put Bertino through a process of multiplying 9 by 40, there was caused an increase in the elevation of his [animatory] cerebral pulse, while the increase in the motion of respiration ensued only after the increase in the volume of the brain had been accomplished." The conclusion of Swedenborg from this fact would be that the mind or the soul is the cause of the motion of the brain by its inflex into it. We shall not attempt to follow him into the regions of philosophy, which are foreign to our present purpose, but we apprehend that there is nothing unscientific in holding the view that the brain-cells have while living the property of self-movement, or, let us say, of motion. Cells in general, when examined under the microscope, even those which have no known or discoverable trace of nervous matter about them, may be seen to shrink and swell, to contract and dilate under the eye of the observer. If cells in general do this, science names the frequent repetition of the phenomenon as *mobility*, or, she sometimes says (it is just here that science is apt to get puzzled, and to use phenomenal expressions as if they applied to things in themselves), *cells have the property of contractility*. Now if in the scientific sense cells have the property of contractility, there is plainly nothing unscientific in Swedenborg's assumption that the respiratory motion of the brain is a property resident in the brain-cells fundamentally, and at least, as we have seen, Mosso's scientific inquiries are in keeping with this conclusion.[1]

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[1] It was during the discussion of this very question, when the question was whether contraction and dilatation might be called the manifestation of the "Cell-soul," that Haeckel on a recent occasion indulged in that wild demonstration against Virchow, accusing his former master of breaking away from the teachings of his earlier days, and by his vehement and almost passionate language reminded us of nothing so much as a heart-broken and despairing lover undeservedly thrown over by the being he had adored. We believe that if Virchow, the scientific evolutionist, had been clearly distinguished from Virchow the philosopher, or spiritual teacher, all of this wild "scene" would have been avoided. But there

Arising out of Swedenborg's views on the self-originated respiratory motion of the brain naturally follows his position concerning the government of the cerebral arteries, which, according to him, are emancipated from the government of the heart, and have their blood-current moved in the stream of the animatory motion of the brain, which is synchronous with the respiration. There are three facts which corroborate the truth of Swedenborg's position, or, we may even say, prove it. First, the brain and cerebellum are supplied by the internal carotids and vertebrals, while the arteries of the dura mater come from the external carotids. Second, the internal carotids and vertebrals undergo a remarkable series of twistings, just before entering the brain, which can have the effect only of breaking the force of the cardiac blood-current, and emancipating the vessels from the heart's impulse. And, thirdly, they lose their muscular coats, and become dilated into various ampullæ which must have the same effect on the circulation contained within them. An examination of such modern writers as Quain, Carpenter and Key and Retzius tends strongly to confirm the truth of Swedenborg's views in this particular.

We propose to leave a consideration of Swedenborg's views as to the glandular structure of the brain for future discussion, as subsequent volumes of this important work appear. Meantime it must be remembered that the theory must be taken as a whole, the particular propositions which we have discussed concerning the motion of the brain being only parts of the whole, and such as, in the view of the author, could be no otherwise than he stated them, and as they were in fact, having regard to the part which the brain plays in the body.

A quotation from Swedenborg himself will fitly bring this review to a conclusion. It places his theory in a harmonious manner before the reader, and also states, what we have made only the most incidental reference to, his view as to the course taken by the animal spirit or fluid secreted by the brain acting in its glandular capacity.

"It is absurd to suppose that any action can begin in the middle of a fibre, and not in its first terminus. If then it begins in the first organs, it must inevitably begin in the cortical glands" (or nerve-cells) "for the fibres commence, and are conceived and produced in those glands, and the arterial vessels of the brain terminate also in them. Hence if the principles of motion exist in them, according to all physical and philosophical laws, as mutually confirmed by and confirming each other, those principles must necessarily commence by a kind of active, living, or locomotive, reciprocal force, that is, by a kind of expansion and constriction, or systole and diastole, such as we observe in a gross form in the lungs and heart; for the same conditions are involved, whether the spirit

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is more to be said than this, strong as it seems, in favour of Swedenborg's views as to the self-origination of the respiratory motion of the brain. Space fails indeed for the full consideration of the question, but we refer the reader who may feel interested in it to Dr. Tafel's note, pp. 670–677, and also to pp. 680–691, where he will find the whole subject historically and exhaustively treated.

is to be driven through the fibres, or the blood through the vessels. The blood cannot be driven through its arteries, without the reciprocal expansion and constriction of the heart; nor can the spirit be driven through the fibres, which are little canals and vessels analogous to the arteries, only more pure, without the reciprocal exhaustion and constriction of the cortical glands of the brain, which, on this account, deserve the appellation of pure corcula or little hearts. Assuming, or granting these points, the necessary consequence is, that every time the cortical and cineritious substance of the cerebrum, cerebellum, medulla oblongata, and spinal cord contracts, or constringes itself, the whole mass of these parts sinks down, and undergoes systole; but, on the other hand, undergoes diastole, when the same substance, I mean the whole congeries, expands. This is the animation of the brain—using the term brain in its widest acceptation—that corresponds to the respiration of the lungs. We must now proceed a step further. If the animal or nervous spirit, at the interval of the constriction of these organic substances—of the little hearts of the cerebrum—is expressed by the cerebrum through the nerves and nervous fibres, of course it is expressed by the cerebellum into its grand sympathetic nerves, the pueumo-gastric nerve, and the great sympathetic cords: and granting this, it follows that these nerves act during the same intervals upon the fibres of the pulmonary plexus, and upon the fibres of the costal nerves; which cannot fail on the instant to act upon their muscles and membranes; nor the latter to act upon the ribs, and thus upon the internal structure of the lungs. Hence it follows that the animation of the brain (using the term here again in its widest sense) must necesarily be coincident with the respiration of the lungs: and the fact is still more plainly declared by the influx of the fibres of the above mentioned cerebellar nerves, the pneumo-gastric, and the great sympathetic, into all the viscera of the abdomen; and by the motions of those viscera agreeing exactly, and keeping perfect time with the respiratory motions of the lungs."

No doubt there is here a mixture of scientific and metaphysical considerations, although the former largely preponderate; but we venture to say that the scientific reader who takes up the book will feel it well worth his while to separate the one from the other, as we have attempted to do, and that he will admit that the method of a writer who so long ago could make contributions to brain-physiology of so permanent a value, must have some qualities that entitle it to attention.

A. Rabagliati.

Critical Digests and Abstracts from Journals.

Bernhardt on Apoplectiform (and Epileptiform) Attacks in Tabes.—Bernhardt (*Archiv f. Psych.* Bd. xiv., p. 135) reports three cases of tabes in which peculiar apoplectiform attacks were noticed. The attacks came on suddenly, and lasted sometimes an hour, sometimes several days. In some instances there was loss of consciousness, in others consciousness was retained, and there was simply aphasia, or loss of power in one limb or in one side. Similar apoplectiform attacks are known to occur in alcoholism, senile dementia, disseminated sclerosis, and general paralysis, but the cases reported showed no symptoms of these diseases. The absence of any of the symptoms of general paralysis is particularly noteworthy, for it is well known that tabes and general paralysis are not unfrequently associated. It is possible, indeed, that in Bernhardt's cases symptoms of general paralysis would have ultimately shown themselves, as five or six years have been known to elapse before the symptoms of general paralysis supervened on those of tabes. But within the last few years Debove, Lecoq, and others of the French school have reported cases of apoplectiform and epileptiform attacks occurring in the course of pure cases of tabes; so that we must recognise these transient attacks of hemiplegia and aphasia as among the symptoms of tabes—often, indeed, as among its earliest symptoms. It is difficult to account for them; but this much is certain—that they do not betoken any serious brain lesion.

Westphal on a Disease resembling Cerebro-spinal Sclerosis.—Westphal (*Archiv f. Psych.* Bd. xiv., p. 87) reports with great fulness two cases which presented the symptoms of multiple cerebro-spinal sclerosis. There was paresis of the muscles of the extremities, the trunk, and the neck, with rigidity of the muscles, and as a result of this delayed and difficult movements; there were tremors when voluntary movements were made; there was "scanning speech" (in one case only, in the other it resembled rather the speech found in bulbar paralysis); the sensory disturbances were slight and transient; the patellar reflex was evocable, and also the paradoxical contraction; and there were headache, vertigo, apoplectic and hemiplegic attacks, and mental symptoms, particularly dementia. The absence of nystagmus would be regarded by some as an important omission from a case of multiple cerebro-spinal sclerosis, but Westhal's experience is that it is frequently absent. In its place there was an abnormality in the movements of the ocular and facial muscles; the muscles responded slowly to the stimulus of volition, and the movements when commenced were slow in execution. This was well seen in the length of time it took to open and close the eyes. Both cases terminated fatally after a very chronic course. In one case a careful examination failed to reveal any cerebro-spinal lesion, in the other the cerebral pia mater was somewhat œdematous, the convolutions small, and the white matter of very firm consistence; but there was not any sclerosis, in the ordinary acceptation of the word. Westphal concludes from these two cases that there is a general neurosis (which, for want of a better name, he calls a pseudo-sclerosis) which cannot be distinguished, either in its symptoms or its course, from the disease known as multiple cerebro-spinal grey degeneration, or sclerosis.

Westhal adds some remarks on the so-called paradoxical contraction of a muscle brought about by its relaxation, the approximation of its points of origin and insertion. It is best seen when the foot is passively flexed; the foot remains flexed and adducted, owing to contraction of the tibialis anticus, and it may continue flexed for half an hour. In one of the cases reported in this paper, the paradoxical contraction was observed not only in the tibialis anticus, but in the flexors of the knee-joint, in the supinator longus of the forearm, in the muscles of the wrist and finger-joints. This condition is due to a change in the muscular tonus, and bears an analogy to Thomson's disease, but we as yet know very little about it.

Senator on Paralysis of the Fifth Nerve.—A man aged thirty-nine (*Archiv f. Psych.* Bd. xiii., p. 590) presented the following symptoms:—Complete anæsthesia of the left half of the face, mouth, and nose; loss of taste in the anterior part of the left side of the tongue; anæsthesia of the left cornea and conjunctiva, there being no blinking on touching the left eye, but if the surface of the right eye was touched the eyelids of both eyes closed, and the ordinary blinking movements of both eyes occurred as in health. Left conjunctiva much injected, cornea softened and opaque, tension of the globe diminished. The only impairment of mobility was paresis of the left masseter. The diagnosis was lesion of the left trigeminus at some point between its exit from the pons and the gasserian ganglion. The patient gradually improved under the use of iodide of potassium, dry cupping, and blistering, but there appeared from time to time slight and transient swelling—first of the left knee-joint, then of the right knee-joint, and afterwards of both ankle-joints.

Senator regards the above as a case of neuro-paralytic keratitis, and makes some observations on this affection. Clinical facts oppose the theory of external irritation as the cause of the phenomenon, and the present case shows that the eyelids on the affected side still afford a considerable protection to the eye. Clinical facts equally oppose the idea that the arrested flow of the lachrymal secretion and the consequent drying of the cornea cause the ophthalmia. The only feasible explanation is that the appearances are due to some influences acting on the nutrition of the tissues, whether through the vaso-motor nerves or through actual trophic nerve fibres is not known.

Senator adduces this case in proof of the old view that the gustatory fibres of the chorda tympani take their origin in the trigeminus. The swelling of the joints he attributes to a vaso-motor or trophic disturbance; it certainly was not a rheumatic affection.

Moeli on the Reaction of the Pupil on Stimulation of the Sensory Nerves. It is well known that stimulation of the sensory nerves causes dilatation of the pupil. Moeli (*Archiv f. Psych.* Bd. xiii., p. 602) has almost never failed to obtain this result in women and children, though in some women over fifty the reaction was not so marked as in younger individuals. In men over fifty, ordinary stimulation failed to evoke it in a number of cases, but powerful electric stimuli generally succeeded. Moeli made observations on the condition of reaction in various nervous diseases. In *general paralysis* he found that where the pupils reacted well to the stimulus of light, sensory stimulation failed to produce dilatation of the pupil in 13 per cent. of the men; there was a decided dilatation in 74 per cent. men, and 100 per cent. women. Where the pupils did not react to light, there was no dilatation in 65 per cent. men, and 50 per cent. women; decided dilatation in 10 per cent. men, and 14 per cent. women. In cases of *tabes* with pupils not reacting to light, dilatation was obtained very rarely, and then it was very slight, and obtained only after strong stimulation. It is interesting to note that in male general paralytics with retained patellar reflex the pupil did not react to light in 36 per cent., whereas in paralytics with absent patellar reflex the pupil did not react to light in as many as 83 per cent. The corresponding figures in women were 40 per cent. and 63 per cent. These figures suggest a connection between disease of the posterior column of the cord and the affection of the pupils. In *epilepsy*, the pupils dilated rapidly after even slight sensory stimulation. The dilatation of the pupil in these cases is not due, as Schiff and others have held, to "an actual cerebral sensation," for it is found during sleep, in coma, and in cases of hysteria with hemianæsthesia after stimulation of the anæsthetic side. And the absence of the pupil reaction must not, especially considering its frequency in tabes, be put down to disease of the cerebral cortex, as is too often done in cases of general paralysis.

Schwarz on the Skin and Tendon Reflexes.—In this paper (*Archiv f. Psych.* Bd. xiii., p. 621), Schwarz endeavours to support, on clinical grounds, the theory that the different spinal centres are related to different brain centres, and that the varying condition of the various spinal reflexes is due to the varying condition of the corresponding brain centres. He points out that where the brain centres are in a state of excitation, as in cases of cortical epilepsy with descending degeneration of the motor tracts (he reports several cases) there is increase of the tendon reflexes, especially directly after the motor discharge; the patellar reflex being generally increased on both sides, the ankle phenomenon being only observable on the side of the convulsions. The skin reflexes are increased, except after very severe convulsions, when they are diminished or absent. In cerebral hemiplegia the tendon reflexes are absent when there is simply paralysis, but heightened if the lesion has produced irritation of the motor strands. The best index of an irritative lesion of the motor strands is the heightening of the tendon reflexes; it often precedes the appearance of contracture by several days. The skin reflexes are always absent or much diminished.

Schwarz distinguishes between the achilles-tendon reflex and the ankle-clonus. He relates a case in which passive dorsal flexion of the foot produced ankle-clonus merely, while percussion of the achilles-tendon produced, in addition to plantar flexion of the foot, general muscular spasms of both legs. The paper concludes with an examination of the various theories that have been offered in explanation of the phenomenon of the tendon-reflex.

De Jonge on the Tumour of the Medulla Oblongata with Diabetes. Mellitus.—In this case (*Archiv f. Psych.* Bd. xiii., p. 658) a tumour the size of a small bean, and consisting of a tubercle caseated in the centre, projected from the left side of the medulla oblongata; it reached from just below the olivary body to the first cervical nerve, and destroyed the substantia gelatinosa of the posterior cornu between these two limits. All other parts of the medulla and cord were intact. The case was one of tuberculosis of the lung and mesenteric glands, in which diabetes was a subsequent and secondary symptom, and the author argues that the tubercle in the medulla oblongata was the cause of the diabetes. He cites eleven cases of diabetes with lesion of the medulla oblongata and neighbouring parts, in all of which the lesion was situated considerably higher than the tubercle in question, and he concludes that the tubercle did not affect the so-called diabetes centre itself, but probably implicated the nerve strands in their course from it to the periphery.

Kirchhoff on Hemiathetosis.—Kirchhoff (*Archiv. f. Psych.* Bd. xiii., p. 582) reports a case of hemiathetosis of the left side appearing suddenly in a patient of intemperate habits, suffering from cardiac dilatation and cirrhosis of the liver with ascites and œdema. The case differed from ordinary cases of athetosis in that the movements were not confined to the hand and foot, but were participated in by the eyes, jaw, and face. After a time slight athetoid movements appeared in the right hand. There were no sensory symptoms, and only transient and inconsiderable paresis of the left hand. The patient died three weeks after the appearance of the hemiathetosis, and a careful *post-mortem* examination failed to show any coarse brain lesion or any miliary apoplexies or embolisms. Delicate false membranes were found in the arachnoid sac of each side, but these Kirchhoff refuses to consider as the cause of the unilateral athetosis.

Flatten on Diabetes Insipidus.—Flatten (*Archiv f. Psych.* Bd. xiii., p. 671) records a case of diabetes insipidus associated with paralysis of the external rectus of the left eye and paresis of the same muscle of the right eye. The diagnosis was a focus of softening a little below the nucleus of the left abducens nerve, destroying the root of the nerve and extending across the middle line towards the right abducens. Gayet has reported a case of diabetes insipidus with paralysis of the right abducens, and Kaemnitz a case of diabetes mellitus with paralysis of the same nerve.

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THE CEREBELLAR CORTEX.[1]

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DR. GAULE, under whom I have worked, and whom I have much to thank for his kind assistance, advised me to use Weigert's säure fuchsin method (*Centrbl. für die med. Wiss.* 1882, p. 753), which has produced excellent results. The first sections were made from pieces of cerebellum, one centim. square, hardened in three per cent. solution of bichromate of potash and kept in a hot chamber at 35° C. for 4–8 days. They were then washed in water half an hour, then placed in alcohol 24 hours, and then for 24–28 hours in a concentrated aqueous solution of säure fuchsin, also at a temperature of 35° C. The pieces were then slightly washed in water and put into strong alcohol and dehydrated, imbedded in paraffin and cut into sections, which were then fixed to object slides, the paraffin dissolved out,[2] and the sections washed out according to Weigert's method with water and alkaline alcohol.

In the second series of preparations they were further stained with nigrosine after the above treatment; and these preparations proved the best. The third series were hardened in the same way, but after fixing the sections on the slides, they were stained by hæmatoxylin and eocené. This latter method of double staining has already been employed by Denissenko. In the fourth method employed, the pieces of cerebellum were hardened by $\frac{1}{4}$ % chromic acid, imbedded unstained, and the sections fixed on the object slide. The specimens were taken from the cerebellum of man (adult and new-born) dog, rat, rabbit, hen and pigeon. My description is chiefly concerned with the dog, in which all the elements are most clearly shown. The human cerebellar cortex is the most developed, and as it is much richer in medullated fibres, and the medullated sheaths are better developed, it would undoubtedly give better preparations, but unfortunately it is impossible to harden the pieces directly after death and so obviate post-mortem changes. And great stress is laid on the fact that in the preparations used for this paper, in less than an hour after death, the pieces were in the hardening fluid in the warm chamber, which was used to introduce a quicker diffusion in the substance of the piece.

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[1] This article is an extract from a paper published in the 'Archiv für Anatomie und Physiologie' (Physiologische Abtheilung) and from work done in Professor Ludwig's Physiological Institute in Leipzig.

[2] Canini, in these Archive, 1883, p. 155.

1.—Granule Layer.

Basis of description, säure fuchsin and nigrosine preparations. In these the medullated fibres are seen, as described in Fig. 1 and 2 *a*, as a black or grey thread enclosed in the medullary sheath stained red. It is very important to note that the axis cylinder is dark and the medullated sheath red. If the säure fuchsin be not sufficiently washed out, other fibres which are not medullated are dyed red, but these have not the dark, central fibre which can be stained by nigrosine or hæmatoxylin, and my description refers only to these fibres.

Fig. 1 is a longitudinal section of a lobule of the cerebellar cortex seen under a low power. Along the centre of this run the medullated fibres, radiating out and passing through the granule layer in the direction of the Purkinje cells. These fibres have been described by Deiters,[1] who maintained the connection of the axis cylinder process of these cells with a medullated fibre. Koschewnikoff[2] has isolated Purkinje's cells in connection with a medullated fibre. Hadlich[3] says that from the white medullated-fibre centre, fibres proceed to the grey red granule layer, divide, and pass as a principal nerve process into the large ganglion cell, into every cell one fibre. Golgi[4] makes the axis cylinder process, in its course through the granule layer, give off branches at right angles. Other descriptions are of a similar nature. But how very indistinctly the fibres have been seen is shown by the very contradictory statements, as for instance some say: that the axis-cylinder runs direct to the medullary centre; that it runs in a horizontal direction; that this axis-cylinder soon gets a medullated sheath, or that this occurs at a great distance from the cell; and that the fibre is fine,—or thick. Nearly all maintain that it divides. Hadlich, and, later, Denissenko make reservations on account of possible deceptions. Hadlich's statements seem to me the most correct.

Fig. 1 shows a clear picture of the whole arrangement of the nerve-fibres; in each lobule some of them can be traced uninterruptedly in their whole course to the immediate vicinity of the Purkinje cells. The fibres leave the medullary centre with the slightest change of direction. At the summit of the lobule they radiate out like the outstretched fingers of the hand. At the lateral part of the lobule they ascend slantingly in a slight curve through the granule layer. They run at definite distances from each other, corresponding to the distances between the Purkinje cells, to which their number, as far as can be ascertained, corresponds. At the bottom of the sulci the fibres are closely aggregated and several pass through the granule layer together.

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[1] Deiters, 'Untersuchungen über Gehirn und Rückenmark.' Herausgegebene v. M. Schultze, 1868.

[2] Koschewnikoff, "Axencylinderfortsatz der Nervenzellen im Kleinhirn des Kalbes;" M. Schultze's 'Archiv,' 1869, Bd. v.

[3] Hadlich, "Mittheilung über den Bau der menschlichen Kleinhirnrinde." 'Arch. für mikr. Anat.,' 1869, Bd. vi.

[4] Golgi, 'Centralbl. für die med. Wiss.' 1879.

These fibres make a bend at the border of the molecular layer, running parallel with it for a short distance (see fig. 1); they do not cross each other and do not branch; but keeping the same thickness, they run straight through the granule layer with some slight wavings in their course. Their axis-cylinder is thick, the medullated sheath slightly varicose, often presenting a jointed appearance. In short, these fibres have in the granule layer the same formation they had in the medullary centre. Each remains an isolated medullated fibre which, unbranched and independently, directs its way towards a particular ganglion cell. One can hardly doubt that these are their nerve fibres. Sections are not very adapted to trace the passage of a fibre into a structure of so much greater size. If the sections are very thin, the chances of finding the right spot are extremely small, and this difficulty is increased as the fibres always change their direction before entering the cell. Isolated preparations show more easily the connection, but then it is not certain that the fibres are of the same nature. I have therefore not been satisfied till I could confirm also in section preparations the connection of the Purkinje cells with one of these fibres.

It can therefore now be said for certain that every Purkinje ganglion cell is in connection with a distinct, isolated, medullated nerve fibre. Besides the above described fibres, there are others, which have already been seen and which caused the different opinions concerning the fibres going to the ganglion cells, as it was not known that there were two different kinds of fibres. Only Hadlich[1] has as yet expressed this idea, and laid stress on the fact that the many divisions described must refer to fibres of the second kind. The second form of fibre produces a plexus which traverses the granule layer in all directions, passing into the medullary centre on one side, and into the molecular layer on the other. These fibres branch about and anastomose, they are of different thickness, from the finest calibre up to that of the first kind of fibres, their medullated sheath is less developed, is always varicose, often interrupted, and sometimes the fuchsine reaction entirely fails. In man these fibres appear far more numerous and better developed than in the dog, when stained by säure fuchsin. Perhaps age makes some difference.

It is quite clear that these fibres differ from those of the first kind. The latter pass through them without being connected with them. Probably all branched fibres belong to the same species and have the same function. I shall designate the direct fibres of the first kind, joining ganglion cells as *straight*, or *unbranched* fibres, those of the second kind as *branched* or *anastomosing* fibres.

The spaces between the fibres in the granule layer are filled up by the closely packed cells. Formerly these cells were all called granules, and their cellular nature was doubted. Denissenko has shown that we have here two kinds of cells, one stained by hæmatoxylin, the other by eocene. The hæmatoxylin cells correspond to those formerly called granules, but they possess a nucleus and protoplasm, and therefore the attributes of a cell. Denissenko regards these as connective tissue cells, whereas the eocene cells are connected with nerve-fibres.[1] The reason why this formation was not recognised before, was because the cells are so tightly packed. Very thin sections have to be stained by a dye which will colour the protoplasm, for this is always present in the form of a very small layer around a relatively large nucleus. The protoplasm has numerous processes, very fine or thicker threads, which form a thick network with the threads of other cells.

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[1] Hadlich, op. cit. p. 20.

The threads refract light strongly and have a sharp contour, the meshes are fine. They are best seen, where the cells lie less packed together, at the borders of the granule-layer. Towards the medullary centre there is no sharply-defined boundary, and as in the granule layer there are everywhere fibres, so in the medullary centre there are similar cells scattered everywhere. But here the protoplasm is more abundant, the threads thicker, and the meshes of the network are very much larger and join with the narrow network inside the granule layer, resembling the fine framework in the white substance of the spinal cord. The nucleus of these cells is very large and characteristic. It possesses a distinct thread-like appearance, with the above-mentioned method of hardening. The threads lie grouped in all kinds of forms in a round clear space. If these nuclei were richer in colouring matter, and if characteristic stages could be found, the idea of nucleus-division might be entertained. Some of the nuclei are certainly larger and their contour more distinct. The mass which is coloured by hæmatoxylin is naturally that of the nucleus threads, while the protoplasm, even when coloured by nigrosine, only appears as a narrow line running round the nuclear spot. Between the cells the medullated fibres make their way, and their medullated sheath comes into most intimate contact with them. The cell threads often seem to penetrate between the separate particles of the medullated sheath. Denissenko describes these cells as grouped round openings from which channels proceed, whose walls are likewise lined by these cells. This appearance is seen when the cells lie particularly close together. These channels are only the medullated fibres which have penetrated between the cells, whilst the place where the fibres cross each other gives rise to the apparent openings. If the cells are less closely arranged, the spaces occupied by these fibres are less compressed, and this presents the appearance of cells in groups surrounded by fibres.

In these spaces other structures are seen, which colour with eocene and nigrosine, and called by Denissenko, *eocene cells*. They seem to be interposed in the course of the medullated fibres, which cross over the cells. They are often really interposed, as can be seen by using nigrosine and säure fuchsin, when the cells become the same colour as the axis cylinder which is then seen to be in connection with them, while the medullated sheath is interrupted.

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[1] Henle and Merkel have already compared the granules to the cells of reticulated connective tissue, and have therefore described them correctly according to their appearance.

Their cellular character is doubtful, as I can find no nucleus in most of them. The larger ones resemble multipolar ganglion cells by their form and size, as also by their connection with nerve fibres; and as it is often difficult to discover a nucleus in ganglion cells when the protoplasm is coloured, these are probably ganglion cells. The smaller of these cells are often deficient in any cellular character and appear to be thickenings along the axis cylinder.

I think I can with certainty explain the use of the hæmatoxylin cells, viz. that of glia-cells. These have not only nuclei, but are cells with protoplasm and processes; the processes form a network which extends through the whole granule layer, and which is also connected with a similar network in the medullary centre.

These appearances are just the criterion of glia-cells, and if one accepts glia-cells at all, they must be cells like those above described. Schwalbe has lately objected to the glia-cells generally, from the hypothesis that the cells can be separated from the fibres of the finest network. But these objections are mostly theoretical. Besides, in my preparations, the network of the fibres proceeds from the body of the cell (see plate Fig. 2, *b*) and this agrees with what Henle and Merkel give in their work on the supporting structure of the central nervous system. Also in the description of the supporting framework of the molecular layer I have only to confirm their observations, agreeing with them that these cells are glia-cells, but doubting that they are cells of reticulative connective tissue, or that they may become ganglion cells. Since the embryologists have shown that the central nervous system proceeds from the ectoderm, and that there exists a thorough separation between the epithelial and connective tissue structures, the "archiblastic and parablastic" formations, it is probable from embryological researches, and what we know of its chemical nature, that the glia is an epithelial formation.

The glia stands in relation to the ganglion cells and their protoplasmic processes, as the medullated sheath does to the axis cylinder of the peripheral fibres. It is a continuation of this latter, and there are forms of transition. The cellular elements are common to both; from this can be explained, what might be considered an objection to the definition of these granule cells as glia-cells, viz., their accumulation into distinct regular layers. Where numerous fibres lose their medullated sheath (those of the first type), or acquire it (those of the second type,) there are to be found accumulated the cellular elements which are common to the glia and the medullated sheath, and where this occurs in regular planes in the cerebellum, the position of the cells is found to correspond.

2.—MOLECULAR LAYER.

a. The ground Substance.—This layer owes its name to the peculiar substance forming its foundation. The older authors describe it as a finely granulated mass, in which a finer structure is not recognisable; however Fromman has already shown it to be a sponge substance built out of a network of threads. Kühne and Ewald have proved that we have here a formation of a peculiar chemical structure, analogous to the horny framework of the medullated sheath of the peripheral nerves. Schwalbe describes the substance in his 'Lehrbuch der Neurologie' as a fine network, the fine trabeculæ being composed of neuro-keratine. This opinion is also borne out by my plates, Fig. 2, *b–d*. The clear substance lying in the enclosed spaces of this network becomes, with the means used, to clear up the medullated substance, as clear as this latter; the osmic-acid method cannot be applied, as the distinction between the trabeculæ and its meshes only comes into view with very thin sections, and these can only be made after treatment with alcohol. The säure fuchsin method, however, produces a very characteristic action on erythrophile substances. In many of the mesh-spaces, but not in all, a fine red line bounds the clear contents, in the same way as seen in the cross-section of nerve-fibres.

These red-bounded spaces are larger or smaller in size, and sometimes several in a row. But they are not sections of fibres, as can be seen from the absence of any axis cylinder; neither are there, where they lie, so many fibres visible, which is easily proved by altering the direction of the section. It thus becomes probable that as this network corresponds to the horny framework of the medullated sheath, so also this intermediate substance corresponds to the myelin; but not chemically, for even the medulla substance of the periphery differs from that of the central nervous system in not re-acting to säure fuchsin; and we must consider that the relation of the neuroglia of the central nervous system differs more from the peripheral nerves than does the medulla of the fibres of the central nervous system. There are, however, many transitions, viz., the above described varicose sheaths of the branched fibres, which often form separate round formations and the glia-nets containing erythrophile substance. Another analogy becomes visible, when we consider the cellular elements of both, viz., the glia and the medullated sheath. Ranvier has acquainted us with the cells of the medullated sheath, and he concludes that the myelin is enclosed in the meshes of their protoplasm. Towards the sheath of Schwann and the axis-cylinder the protoplasm is thickened to form the inner and outer protoplasmic sheath. When this theory was proposed, Kühne and Ewald had not yet discovered their horny sheath, which probably corresponds to Ranvier's protoplasmic sheath, if neuro-keratine be substituted for protoplasm. I think it probable that from the protoplasm of the young cell, neuro-keratine is formed by differentiation, as is similarly described by Waldeyer in the horny change of the epidermis cells. There, a stroma of keratine threads forms itself out of the protoplasm of the cell, while in its meshes, a fatty substance, eleidin, is deposited. It is difficult to distinguish between the sheath and the framework. Either can be resolved into neuro-keratine. Probably transitions between the two will be found. I have also tried to convince myself in preparations of the horny sheath, treated by Kühne's method and coloured by hæmatoxylin and nigrosine, whether the neuro-keratine threads really are in connection with the nuclei of the medullated sheaths, and I can assert that one can really see this, so far as it can be observed. Assuming the horny framework of the peripheral nervous system to be a formation of cells, its relation to that of the central nervous system appears all the more assured. For not only the network of the granule layer already described, but also the network of the molecular layer, appears as glia-cells. The connection between both is best observed in the collection of glia-cells to be found at the base of the molecular layer. When from any reason, the molecular and granule layers are separated—which may be caused even by pressure of the cover-glass—there remains one or more layers of cells separated with the molecular layer. The separation occurs below the Purkinje cells, where the unbranched fibres bend round and spread out. Here, the layer of glia-cells attached to the molecular layer forms a membrane, by joining their processes, to support both the fibres and blood-vessels spread out under it, and the Purkinje cells resting on it, and to form also the groundwork of the molecular substance. In Fig. 2 *b*, this is shown in section. Its cells are generally larger than those of the granule layer, as are also their protoplasm and processes, which here become very distinct. Many are pyramidal or pear-shaped, with their broader basis turned to the granule layer, and form, with the long processes given off from the glia-nets of the granule layer, that membrane of the molecular layer which may be represented as a flat network broken up by the cells interspersed in its meshes. Through this membrane pass all the numerous formations on their way from the granule to the molecular layer, and vice versâ. As the name of *limitans externa* has been adopted by Henle and Merkel for the layer of the glia-nets which is in contact with the pia mater, and this layer, by its formation and origin being completely analogous to the above-mentioned membrane on the inner surface of the molecular layer, I propose to call the latter *limitans interna.* Some of the processes from its cells, which are vertical to the limitans interna and go off from the apex of the cells, are particularly thick and can be traced far into the glia-network; and at regular intervals these processes are still thicker and reach to the limitans externa, to which they are attached by a flattened pedicle and serve as supporting pillars to the molecular layer. These were discovered by Bergmann and described by Henle and Merkel. They have also described the limitans externa as a special membrane, separable from the pia mater, and consisting of glia threads. Obersteiner has described one or two cell layers under the pia mater in the new-born animal, from which layers is developed the limitans externa, as well as the supporting pillars; probably the limitans externa has the same character in the new-born as the limitans interna in the adult, so that the pedicles are remains of pyramidal cells, whose apex processes formed supporting pillars and basal processes the network of the limitans externa. The network between the membrane is also probably the remains of the cells described by Obersteiner, as filling up the embryonal molecular layer, and which have been absorbed with the growth of the network; as the network is too extended to have been formed from the supporting fibres, or from the glia-cells scattered in the molecular layer. Perhaps the protoplasm of the embryonal cells may have been converted into neuro-keratine. Two further points about the glia-cells remain to be discussed. (1) Their relation to the ganglion cells and their processes; (2) their relation to the connective tissue.

(1) We have already mentioned that the glia-cells are accumulated round the ganglion cells (see Fig. 2 *b*). Whilst the ganglion cells at their lower part rest on the limitans interna, they are packed at the sides by these heaped up glia-cells, so that the cells, together with the limiting membrane, closely surround a hollow in which the ganglion cell lies. At the upper and lower part the processes pass out. This walling-in is seen best when the Purkinje cell has been detached from its space, which is then seen spun over with a network of fine fibres, which are continued at the sides into the bodies of the glia-cells. This capsule is thus formed by a net of neuro-keratine threads, supported by a denser accumulation of cells similar to that found in the limitans interna. A somewhat similar capsule has been described by Denissenko and by former authors. Of course such a capsule consisting of a network will allow the cell a free connection on all sides; there is, however, a special condition for the axis cylinder and the protoplasmic processes. The former seems to acquire a continuation of this capsule as a sheath, which is connected with the medullated sheath of the nerve-fibre. But I have no definite drawings on this point. As is evident from Fig. 2, *b*, *c*, everywhere in the course of the protoplasmic processes the glia-nets surrounding them become thickened, so that numerous threads seem to be attached to them, whilst on each side,' the meshes of the glia-nets become larger. This attachment of glia-threads has been described by Hadlich and other authors, who state that the protoplasmic processes appear rough, or that extremely fine threads go off from them laterally.

This attachment is however only apparent; it is in reality only a tube-like thickening of the network around these processes, forming a sheath of neuro-keratine threads, continuous with the capsule of the ganglion cells. This appearance resembles the axis-cylinder of the peripheral nerve fibre (when treated according to Kühne's method), which is closely surrounded by a thickening of the horny structure, decribed by Kühne as the inner horny sheath, and here the keratine threads often seem to join the axis-cylinder itself. The broad meshes of the glia-nets on the sides of the protoplasm processes correspond to the space between the inner and outer horny sheaths, which is also traversed by a few threads. A formation analogous to the outer horny sheath is absent in the glia. The resemblance between the relation of the protoplasmic process to its glia, and the relation of the axis-cylinder to its horny sheath helps very much the comprehension of the central nervous system. But the sheaths in the glia are not isolated, they are in connection everywhere, and are only interrupted by the branching out of the processes. In the glia, the horny component part is unduly in excess of the thread network, as compared with the fatty part enclosed in the meshes: and the fatty substance is not identical with myelin of the peripheral nerves. As Weigert's reaction is only sometimes shown, probably different gradations of myeline exist in the nerve-fibres of the periphery and in those of the central nervous system, where varicose fibres occur, increasing as they become finer.

(2) It is not easy to establish the boundary between the glia and the connective tissue. When they were still unconditionally classed together, it was observed that there by no means existed an immediate connection between the pia and the glia lying under it. Henle and Merkel—who in their treatise assume that glia, nerve elements and connective tissue are produced out of the same materials—describe, however, that the glia limits itself by a lamina limitans externa, and that this is easily detached from the pia, being separated by spaces traversed by single threads,—subarachnoid lymph spaces. A retraction of the glia from the connective tissue sheaths of the vessels leads to the production of the peri-vascular spaces. The glia therefore is only in connection at single points with the connective tissue, which is necessary to give support to the whole framework. In short there seem to be connecting-fibres only between the pia and the limitans externa, as these fibres do not sink deeper into the glia-network.

The connective tissue of the pia may be compared with the Schwann sheath of the peripheral nerves, if the glia be compared with the medullated sheath; and as between the Schwann primitive sheath, and the medullated sheath, so between the connective tissue and the glia, there exists only a loose connection; from this it does not follow that the glia might not histologically be reckoned with connective tissue. But this depends entirely on whether or no the glia-cells are descended from epithelium cells of the ectoderm, for our present classification of the tissue is essentially generic. From the above, it is no longer possible to treat of the glia as an undefined boundary between nervous and connective tissue. We have then in the nervous system three distinct systems lying close together:

1. Axis cylinders.	Ganglion cells.	Protoplasm processes.
2. Medullated sheaths.	Glia-cells.	Glia net-works.
3. Schwann's sheath.	Pia mater.	Connective tissue-sheath.

Of these systems the first only can be designated as nervous.

b. The Nervous elements.—One link in the chain of this nervous system fails us. The branched processes of the Purkinje cells must enter again into connection with nerve fibres. It is easy to trace the branches to close to the limitans externa, but here every direct trace is lost. Rindfleisch and Stricker suppose them to become lost in the ground-substance of the neuroglia, but this has been very much contested, as it is against the physiological demand for a definite course. In my preparations I find no indication of a connection of the ganglion-cell processes with the threads of the glia network. A second view makes the processes end in peculiar nuclei, which lie at the periphery of the molecular layer. I have found it difficult to discover what form of cells Denissenko means by these peripheral nuclei, for at the limitans externa or towards the inner part of the periphery, there are very few cells to be found in the adult animal. Numerous cells are present in the young animal only. Denissenko calls these cells nuclei, and yet states that they are very little coloured by nuclei staining fluids, and further, that in some animals, fibres spring from them which penetrate vertically the molecular layer. The latter fibres correspond to the supporting fibres of the glia, and I consider that the so-called peripheral nuclei are the pedicles with which the supporting fibres attach themselves to the limitans externa. The degree of coloration mentioned by Denissenko agrees also with these pedicles, which might also be called nuclei, as they are possibly the remains of cells; but with the processes of the Purkinje cells these pedicles are not connected.

This view of Denissenko agrees with Golgi's, who makes the processes of the ganglion cells end in connective tissue corpuscles at the periphery, which Denissenko even conjectures to be partly identical with his peripheral nuclei. With the exception of the sheaths of the vessels, I must decidedly dispute the presence of real connective tissue corpuscles at the periphery of the molecular substance on the inner side of the limitans externa. A third view makes the processes of the periphery bend round and run back to the granule layer; and to prove this, certain fine straight fibres, which traverse the molecular layer vertically to the periphery, have been pointed out as these processes (Boll); but the greater number of these fibres can be traced to the limitans externa where they end in pedicles. They are the supporting fibres, as others have already shown.

Lastly, Hadlich has suggested that the ganglion cell processes return again in the same form. This appears at first difficult to contradict, as the processes may be either running out or returning. But Hadlich has already conjectured that there might be two kinds of nerve-fibres, one entering into connection with the axis cylinder of Purkinje's cells, the other with the already mentioned returning branched processes. The latter therefore would not return to ganglion cells, but join directly with nerve fibres. In this way we have a distinct criterion by which to recognise the out-going from the in-going fibres, and a proof of whether they really exist. I have looked through my preparations and found that there are numerous places where, in the deeper parts of the molecular layer, all ganglion cell processes can be traced to their corresponding Purkinje cells. This precludes the possibility that the protoplasmic processes, after spreading themselves out at the periphery, should collect together and return in the same form to the granule layer, to join with nerve-fibres. Another hypothesis assumes that the returning processes reassemble in the ganglion cells, of which there are two kinds, and which are not to be distinguished in their outward appearance. But this is contradicted by the fact that the branched medullated fibres can be traced far into the molecular layer. The object of these fibres could not be seen, if the protoplasmic processes return to a ganglion cell lying at the border of the granule layer before joining the nerve fibres. Besides this the number of unbranched fibres is probably just as large as that of the ganglion cells, so that we can drop the hypothesis of two kinds of ganglion cells. If the number of the unbranched fibres is just as many as the Purkinje cells, and if they end at the granule layer, whilst the branched fibres extend into the molecular layer, the probability becomes very great that this cell is in connection with both kinds of fibres, with those in the granule layer by its axis cylinder process, with the others in the molecular by its branched protoplasmic processes.

Fig. 3 is drawn from a section cut parallel to the surface, and therefore at right angles to the direction of sections hitherto shown. The drawing represents the bottom of a sulcus, so that not only the molecular layer, but also the Purkinje cells and a portion of the granule layer are visible on each side. The medullated fibres appear on the flat surface of the section, running in two directions at right angles to each other, and round the Purkinje cells are numerous fibres passing into the molecular layer. The section here given is very thin, or else the fibres would be very numerous, as is the case in thick sections, where it is difficult to see individual fibres. This horizontal plexus is most dense in the deeper parts of the molecular layer, around and immediately above the Purkinje cells. But also in the more superficial planes, fibres running in the same direction can be seen, and the better the reaction has succeeded, the further does their region extend. They are all much finer than the unbranched fibres, but of the same calibre as the finer branched fibres. In the corresponding vertical sections of the lobule, fibres are seen in bundles close round the capsule of the Purkinje cells and passing into the molecular layer. Little of the above described horizontal plexus can be seen in these vertical sections, as the one set of fibres is cut across (compare Fig. 2, *b*), while the other runs parallel to the surface. On the other hand there are fibres visible, which descend vertically in the third plane. These are also fine and often run close to the supporting fibres. Generally these cannot be traced further than through half the molecular layer. Beyond that, although erythrophile substance is met with, it is not arranged in the form of fibre sheaths, but in isolated rings, without any connection. Probably these single rings must stand in some relation to the continuation of the system of medullated fibres.

As the connection of the fibres of the molecular layer with the fibres of the second kind in the granule layer is very distinct, we may say that there exists here a fibre system, which, starting as a plexus from the medullary centre, branches through the granule layer, until the finest fibres are lost in the molecular layer, whilst their medullated substance can be traced further than the axis-cylinder.

As regards the connection of the separate elements it is easy to establish the following points of a scheme:

1. *Each unbranched fibre is connected with each Purkinje cell. The axis-cylinder passes into the protoplasm of the cell, the medullated sheath into the glia-like capsule of the cell.*

2. *The axis-cylinder becomes converted into a number of fibrils in the cell, which pass into the branched protoplasmic process. The fibrils run in the process, which is surrounded by a glia-sheath, as completely distinct threads as far as the periphery. In the branching of this process, the numerous fibrils, contained in it where it leaves the cell, are gradually distributed until they become isolated.*

My preparations show that the arrangement of the ganglion cells corresponds to M. Schultze's scheme. Obersteiner has already described this for the Purkinje cells. Sometimes fibrils cross each other at the points where the processes branch.

3. *The fibrils thus isolated bend round at an angle of* 90° (*not* 180°), *spread themselves out in a plane lying parallel to the surface, and rearranging themselves in definite order as fibres surrounded by a medullated sheath, run, with frequent interchange between the fibres, in the form of a plexus back to the medullary centre.*

This third point of the scheme contains, of course, much that is hypothetical. But there can be little doubt that the fibres of the second system serve to establish somehow a connection with the branched processes of the ganglion cells. To explain why the fibres should not bend at an angle of 180°, instead of 90°, I must refer to Obersteiner's discovery that the protoplasmic processes of the Purkinje cells only spread out in a plane at right angles to the surface, and also to the direction of the lobule and the plane of the medullated fibre layer. On the other hand, the medullated fibres of the molecular layer run in planes parallel to the surface, and at right angles to the planes of the processes. The processes only need now to bend round at an angle of 90° to pass into the plane of the fibres. Certainly other fibres are required which shall turn at an angle of 90° and connect the higher lying planes with the lower, but these fibres would be coarser and medullated. The strictly angular arrangement of the elements of the cerebellum is remarkable, both in the nervous elements and in the supporting structure. The important point is that we do not see the connecting elements between the ends of the protoplasmic processes and the beginning of the nerve fibres; and in the absence of any really decisive result, some other solution of the problem is possible. Perhaps future research will reveal that just at that point where the continuity of the elements is lost, an arrangement intervenes which we cannot now recognise.

Further, there are medullated fibres under the pia mater lying on the limitans externa, broad fibres of a similar character to those of the medullary centre.

The same fibres may be seen passing through the molecular layer in a slanting direction. A second collection of medullated fibres of the same character lies half-way up the molecular substance. Both run parallel to the medullary centre. As these fibres are

Fig. 1

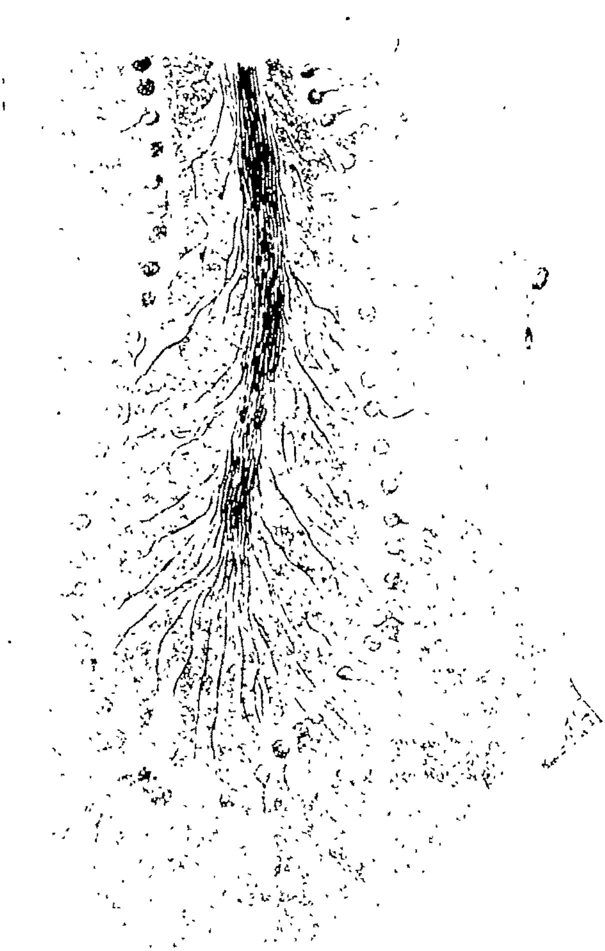


Fig. 2 a

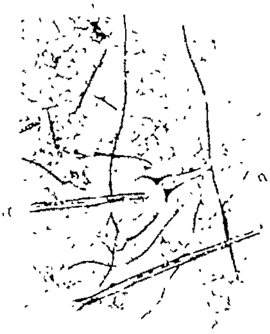
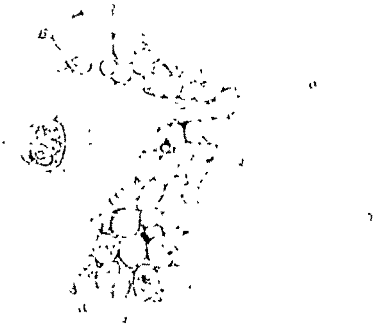


Fig. 2 b.



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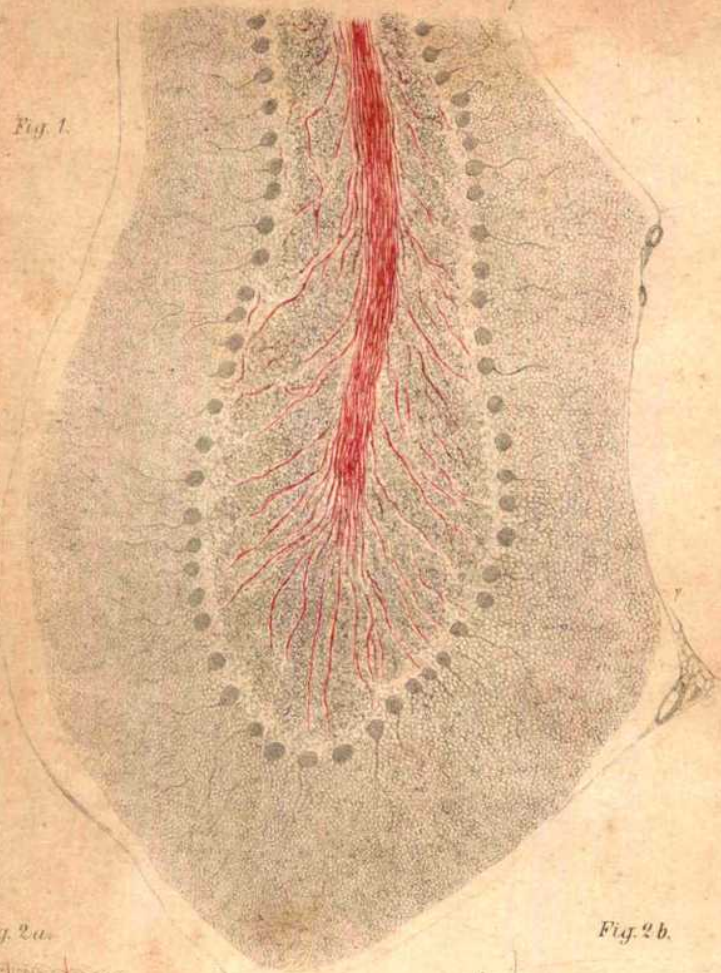


Fig. 2a.

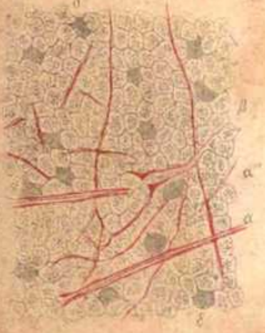
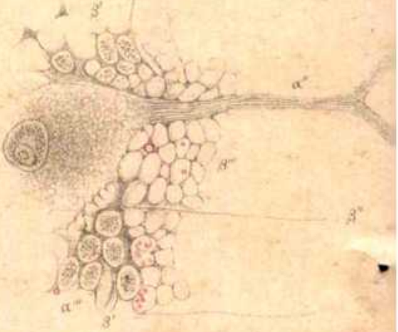


Fig. 2b.



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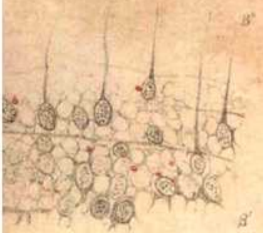


Fig. 2b¹.

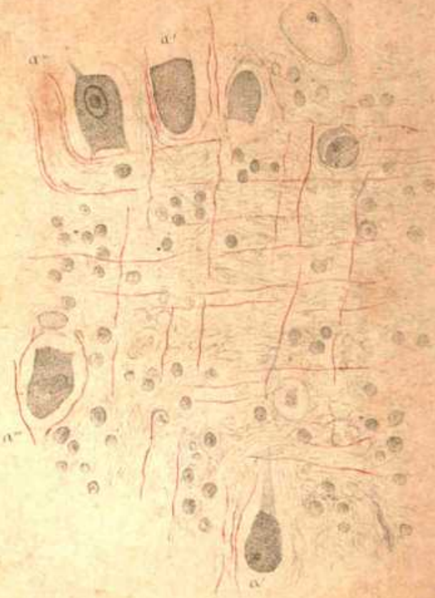
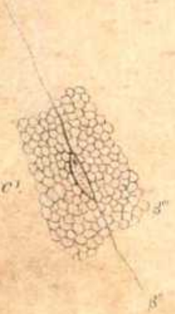


Fig. 3.

Fig. 2c¹.



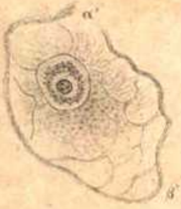


Fig. 2b².

Fig. 2d.

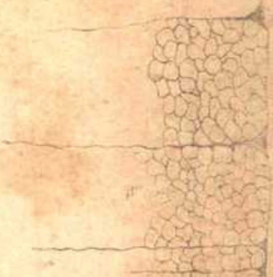
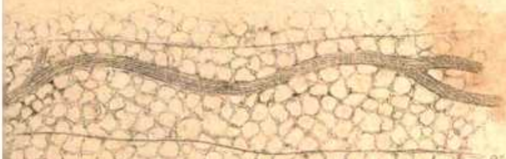


Fig. 2c.



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only found in a small minority of the lobules, they are probably an aberration of the medullary centre.

Sometimes the Purkinje cells of the same lobule differ in appearance. Some show, as seen in Fig. 2 *b*, a distinct nucleus, with the nucleolar contents surrounded by a ring of nuclear substance, and in the protoplasm, a distinct fibrillous formation which completely fills up the glia-capsule and in the double-staining with nigrosine and säure fuchsin are stained a light grey colour by the former only.

In the other kind a nucleus is hardly to be distinguished, and no fibril-formation; the cell appears as a homogeneous body which remains dyed by säure fuchsin, even after washing out by alkaline alcohol. The cells and their processes are slightly retracted from the glia, and the relation between the capsule and its sheath is not quite so distinct. It is not probable that there are here two kinds of ganglion cells, as the difference in the cells is only seen in some of the lobules. It is hardly due either to different action of the hardening re-agent or to the fact of one cell dying before the other, as an external influence would act differently on the cells of two distant lobules, but not on those of the same lobule. We must conclude that the condition of cells must have originally been different at the moment of death.

EXPLANATION OF THE PLATES.

α Unbranched broad nerve-fibres of the first kind.
α' Purkinje's cells.
α'' Their protoplasmic processes.
α''' Branched fibres of the second kind.
β Glia-cells of the granule layer (granules, glia-cells).
β' Cells of the membrana limitans interna.
β'' Supporting fibres (long radiating processes of the glia-cells).
β''' Glia network (short branched processes of the glia-cells).
β'''' Membrana limitans externa.
γ Pia Mater.
δ Non-nucleated structures of the granule layer, stained by Eocene and Nigrosin (Eocene-cells).

GENERAL VIEW MAGNIFIED WITH A LOW POWER.

FIG. 1. Longitudinal section of a lobule from the cerebellar cortex. Medullary centre—internal or granule layer—external or molecular layer. The pia mater with its blood-vessels is slightly separated. Shows the radiation of the unbranched fibres from the medullary-centre through the granule layer to the Purkinje's cells.

FIG. 2. Details of the same section seen by a higher power ($\frac{1}{18}$ oil immersion) showing:

a. Granule layer.
b. Boundary between granule and molecular layers.
c. Middle of the molecular layer.
d. Outer part of the molecular layer.

The figs. 2 *a–d* are to be taken in series.

2. *a*, shows fibres of the first kind, α, and of the second kind α''' between the glia-cells, β, and the eocene cells, δ.

2 *b*. A Purkinje's cell, α' with its protoplasmic process, α'' which breaks up into fibrils. The glia-cells of the lamina interna, β', some of them large and rich in protoplasm, are heaped around the Purkinje's cell. The glia network β''' proceeds from the processes of the glia-cells, some supporting fibres β'' are here not seen joining the cells. In the meshes of the glia-nets are seen sections of medullated fibres of the second kind, α''', stained red.

2 b^1. The glia-cells, in the interval between two Purkinje's cells, form by their intimate connection the limitans interna, β'. Some pyramidal-shaped glia-cells are elongated into processes which extend direct to the periphery and form supporting fibres β''.

2 b^2. A Purkinje's cell in the glia capsule. The cell is seen, so as to show clearly its capsule. The threads of the glia are partly visible coursing over the cell.

2 *c*. Glia network in the middle of the molecular layer. Very clear broad trabeculæ of the network, β''', which is traversed by parallel supporting-fibres β'', and also by a branching process from a Purkinje's cell, showing fibrils, α''.

2 c^1. A supporting fibre, β'', in connection with one of the solitary glia-cells found in this region.

2 *d*. The periphery of the lobule, with the limitans externa β''' cut across at right angles, and with the supporting fibres, β'', attached to it by broad pedicles; between them is the glia network.

FIG. 3. Horizontal section (parallel to the surface) through a lobule of the cerebellar cortex near the boundary between the molecular and granule layers, which is depicted at the periphery of the drawing, so that the Purkinje's cells appear surrounded by medullated fibres of the second kind which having reached the molecular layer, spread out in the plane of the section, in two directions at right angles to each other.

These plates are faithful representations of the preparations, and are not in any way schematic.

BRAIN.

JANUARY, 1884.

Original Articles.

THE RELATIONS OF HYSTERIA WITH THE SCROFULOUS AND THE TUBERCULAR DIATHESIS.

BY J. GRASSET, M.D.,

Professor of Medicine in the Faculty of Montpellier.

THE numerous labours which have of late so successfully been directed towards the transformation and completion of neuropathology have all been inspired by an analogous tendency, and have left aside an important aspect of the question.

The symptomatic pictures presented by the various diseases of the nervous system have been especially determined, and thus distinct morbid types have been clearly established. The physiological pathology and pathogeny of these various clinical syndromata have also been unfolded; finally, in many cases, the anatomical lesion upon which the disease depended has been carefully analysed.

It is evident that such was the right method in unravelling the ancient chaos of nervous diseases, and it is already a great service rendered to the physician to establish definitively for him the symptomatology, physiology, and pathological anatomy of the diseases with which he has to grapple.

But beyond this sphere of knowledge there exists a whole group of facts, the knowledge of which is urgently needed by him to complete his diagnosis and establish his therapeusis: it is the etiology. This chapter has yet to be written for the nervous diseases.

In order that an etiology be living, as it were, complete and clinical, it is not sufficient to indicate the immediate cause which has determined this or that accident, such as a more or less violent emotion, as the beginning of a neurosis, or some sexual excesses as the starting-point of a tabes. Unless we go beyond this proximate etiological element we shall create but a trivial neuropathology, and one without practical applications.

This proposition, which has been at all times as the by-word of clinical medicine, will be still more readily at a time like the present, when even acute pneumonia is considered by many as a localised general disease, and when traumatisms—these most typical of local states—are so visibly influenced and dominated by the constitution, or general habit, of the patient. Most physicians recognise nowadays that the local or physical diagnosis in a pulmonary or cardiac disease, important as it certainly is, remains much below what is needed for a complete and practical knowledge of the disease; that it must be completed by a diagnosis of "nature," by an analysis of the general morbid state which governs this lesion, already well defined by our senses armed with the requisite instruments. If this view is true of the circulatory and respiratory system, it is all the more imperative to take it into account with reference to the nervous system.

Placed in presence of a nervous case, the physician must evidently begin with the physiological diagnosis, that is, carefully analyse the symptoms and connect them with the normal functions of the nervous system; he next proceeds to the anatomical diagnosis, determining the presence or absence of a lesion, its position and extent. This part of the problem solved, and all late researches have tended to facilitate this solution, he will be able to label his patient as tabes dorsalis, hysteria, or cerebral softening; but he will not have reached the end of his duty.

There still remains the *nosological* diagnosis; that is, the determination of the general morbid state, of the diathesis of the constitutional disorder which stands behind this lesion or this neurosis. Upon its establishment depends the institution of a complete and rational treatment. Many physicians accept these views to a certain extent in the case of syphilis, and give their patients the advantage of an antidiathesic medication—but many others decline to entertain them, even with reference to syphilis. At any rate, they are not usually extended to other diatheses, which for us is a mistake.

The fact that we have not as powerful means against rheumatism, scrofula, and other diatheses, as against syphilis, does not imply that we stand disarmed in presence of them. There is an anti-diathesic indication to be fulfilled, and for the fruitful administration of iodine or alkalies it is not indifferent to know whether the disease ascertained is of rheumatic or scrofulous nature.

Neuropathology then requires, like the other branches of pathology, to be completed and enlightened by a thorough nosological study of the causes of the general morbid states.

This preamble was necessary to justify our attempt to study the relationship of hysteria with the scrofulous and tubercular diathesis;[1] and to write, so to speak, a paragraph of this chapter of neuropathology.

Hysteria is a disease of such complexity, tenacity, and peculiarity, that it is often usually considered as a true morbid state, distinct from and irreducible to other disorders. Such is not our view.

Hysteria is a neurosis; but we scarcely can admit of a neurosis except as symptomatic. They point to a peculiar condition of the nervous system which is unknown to us, but which exists—dynamically or anatomically; and this state of the nervous system is in its turn symptomatic of a general disease, or more usually of a diathesis.

Just as chorea is often a manifestation of the rheumatic, and angina pectoris one of the gouty diathesis, thus hysteria is often a manifestation of various diatheses, more particularly of scrofulosis and tuberculosis. Hence when we speak of the relations of hysteria to these diatheses we do not mean that hysterical subjects have frequently tubercles in the lungs, or that the confusion between them and the scrofulous be more or less easy—this is not at all the aspect of the question we are contemplating.

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[1] It is chiefly tuberculosis which we have in view in this paper: scrofula is accidentally mentioned, in consequence of the indications presented by the two diatheses in certain clinical cases.

We mean simply that these diatheses are constitutional states found in a series of generations of the same family, and that among a certain number of subjects of the series they may manifest themselves as neuroses; that especially in certain individuals, children of tuberculous or scrofulous parents, the tuberculosis or scrofulosis may be represented by hysteria only.

In order to grasp this view, we must admit that the diseases known as tuberculosis and scrofulosis are of a diathesic, constitutional nature, frequently manifested by the anatomical lesion designated as tubercular or scrofulous, but not necessarily linked to such lesions.

Tubercles, for me, are not necessary and uniform characteristics of the tubercular diathesis; they occur without it (impaludism, scrofulosis, alcoholism), and there are cases of tuberculosis without tubercles. I cannot develop this proposition here; but I consider it as a demonstrated clinical truth.[1]

Rheumatism may be manifested by chorea, gout by angina pectoris; that is, lesions of the serous or fibrous tissue do not necessarily occur among all rheumatic subjects, nor the uric lesion among all the gouty. In other words, diatheses are not manifested by this or that lesion; they are indissolubly linked to no single anatomical lesion special to them. They may become apparent as neuroses, that is, by syndromata, of which the lesion is not appreciable to our senses.

What we are now saying of rheumatism and gout is true of syphilis and the other diatheses, such as scrofulosis and tuberculosis. It was in order to avoid the similarity between the words "tubercle" and "tuberculosis" that we proposed to call the latter "Laennec's disease," a name which predicates nothing of the anatomical substratum of the disorder, and is therefore not in contradiction with the view we support.

When therefore we say that tuberculosis may become manifested as hysteria, we do not in any way imply that there are tubercles among hysterical people. It is not the coincidence of hysteria with pulmonary or other tubercles which we want here to place into light. We want to show that hysteria may be, and often is, a manifestation of the tubercular diathesis; and two cases may present themselves here according as the neurosis will be the only manifestation of the diathesis, or will be continued in the same subject along with other diathesic manifestations, such manifestations bearing to one another certain relations to be studied.

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[1] See our article *Diathèse*, in 'Diction. Encycl. des Sci. Méd.'

How is the clinical demonstration of such a proposition to be made?

I must say at once that it is not by the direct examination of the patient; it is by the whole pathological history of his life, ascending into its remotest origins; it is by the history of the subject and his family, by the study of the transformation of the disease through generations.

By thus working up into the morbid antecedents of this subject, by following for a long time the ulterior phases of his disease, by analysing the diseases of his ascendants, collaterals, and descendants, one is struck by the relations of intimate affiliation between hysteria and certain diatheses, especially tuberculosis and scrofulosis.

Thus, for instance, in a phthisical family you will see among the children of a tubercular subject one dying of tubercular meningitis, another become an ordinary consumptive, the third seem to escape the diathesis. The last may be neurotic, hypochondriac, hysterical, or lunatic even; but for me he has escaped the hereditary diathesis in appearance only. He is tubercular like the rest, though he has no tubercles anywhere. It is his neurosis which is the manifestation of his diathesic affection. And what proves it is that in his children the diathesis is still present, and will often reassume its classical type. Son and father of phthisical subjects, he does not interrupt the diathesic chain; only pulmonary phthisis is in him replaced by hysteria. Moreover, he may prove the nature of his neurosis by becoming himself phthisical in the second portion of his life; and then there are often most remarkable phenomena of alternation and equilibration between the two manifestations of the same diathesis, phenomena which have caused the supposition of an antagonism between hysteria and tuberculosis, when they were merely successive and different manifestations, mutually replacing one another, of the same fundamental diathesis.

This view will shock certain minds at first sight. It however results from the very nature of things, and is not without analogy with other categories of clinical facts.

How often one sees the descendant of rheumatic parents seized with chorea without having previously presented any articular pains! This chorea is truly rheumatic, as will become manifest by the apparition of articular disorder in himself or in his children. No one hesitates in such a case to frame such a diagnosis. The same considerations obtain when we deal with migraine, asthma, and arthritism, and of all this group of clinical syndromata, of which Bouchard has so well brought the intimate relationship into light in his recent work on diseases from slackening of the nutritive processes.

This relation which we wish to establish between hysteria and certain diatheses does not therefore contradict clinical experience, and must not appear *à priori* irrational. Now it is easy to see the practical importance of this kind of study. The consideration of the diathesis standing behind hysteria, and, generally speaking, behind all neuroses, is not a theoretical notion of pure pathology, but a real clinical datum, yielding indications for the therapeusis.

When one reads the recent important works on nervous diseases, one is infallibly struck at the scantiness of the therapeutical chapters. In the case of hysteria, in particular, one multiplies indeed the means of treating the attack, of removing the anæsthesia; single symptoms are pretty well met and corrected. But the groundwork itself, the neurosis, the morbid state? How powerless we seem in many cases! One scarcely attempts, even, to establish a rational mode of handling the factor which keeps under its dependence all the ever-changing scene of manifestations.

And yet it is there that a new and fruitful path lies. As we shall try to establish it later on, the treatment of the manifestation is often useful; the treatment of the nervous system is indispensable; but more than all this is wanted.

The deeper organic cause of all these nervous derangements, the morbid state which is the true disease, has to be grappled with. We only indicate here the ideas which the remainder of this paper will develop and demonstrate. We shall best return to them after giving the facts upon which they rest. But it is indispensable to put forth synthetically at the very outset the doctrine which we wish to enforce. The reading of the cases will thus be more profitable.

We shall not enter upon the history of the question, which would indeed be very short, authors having scarcely yet considered and discussed this view of hysteria—and this, for several reasons. Firstly, not thinking of the relationships, they have not looked for them—and these are things to be sought. Further, such researches are almost impossible with the material supplied by hospitals. The patients who go there come from unknown quarters; we are debarred from every accurate investigation into their heredity. It is only in private practice, where one has an extensive knowledge of one's patients, of their ascendants and descendants, of their whole family, that one can frame such a nosological diagnosis.

The causes of hysteria in hospital patients are also usually too complex, and one may say too external, to allow the putting into clear relief the pathogenetic rôle of a diathesis. Why seek tuberculosis among the ascendants of a Saltpêtrière patient? Her whole life is insufficient to account for the development of the neurosis; whilst among the upper classes, if you find hysteria in a young woman, or even a child, in a family with a calm existence, with regular habits, and no moral over-driving, you will seek more attentively for the hereditary constitutional vice which has struck this patient, and you will easily discover the diathesis, of which the inportance becomes immense.

The view which I defend here has therefore been frequently indicated to me by my master and colleague, Professor Combal, who, in his immense private practice, continually finds proofs of the intimate relationship between the neuroses and the diatheses. In the first edition of my work on the Diseases of the Nervous System (1879) I said (vol. ii. p. 555): "The general states may also give rise to hysteria . . . diatheses are found in some cases, and would be oftener found if better looked for. Thus one often talks of hysteria among the children of a consumptive subject, and rightly. Hysteria is in such the manifestation of the hereditary diathesis, and cases are seen in which this diathesis manifests itself at first only by nervous disturbances, and later by the typical lesions of the lungs, whilst at the same time one often sees the hysterical phenomena diminish and disappear."

In the second edition (1881), I reproduced this quotation (p. 924), and added (p. 1040): "Finally the tubercular diathesis may betray itself by neuroses, hysteria especially, chorea, &c. I have recently seen two cases in which the hysteria seemed to be the manifestation of the tuberculosis. These facts have a great clinical importance, because frequently hysteria is developed in a young woman with a diathesic heredity; the neurosis is treated; it disappears, and then pulmonary accidents, sometimes of the utmost severity, are evolved. Or the hysterical phenomena may persist during the phthisical process, and present a series of compensatory rises and falls. Finally, hysteria may be the only expression of the diathesis, and phthisis reappear only in the succeeding generation." And I quoted in a note an observation from the "Iconographie photographique de la Salpêtrière," and two from the 'Thesis' of Dr. Paris, which will be mentioned again.

The following authors, also of the Montpellier School, have published papers which may be taken in connection with our present subject: Brousse,[1] Largaud,[2] and Mossé.[3]

Most classical authors either say nothing on the subject, or mention our doctrine only to set it aside. Thus in his recent 'Traité des Névroses,' Huchard says (p. 1056): "Some authors have made a mistake in saying that tuberculosis gives rise to hysteria:" further (p. 1073): "Among the diathesic diseases, phthisis has been quoted among the antecedents of hysteria. However, it is not the latter morbid state which is to be incriminated, but only the arthritic diathesis which may give rise to both tuberculosis and hysteria, and the latter is not in any way depending upon the tubercular affection."

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[1] 'Gazette des Sci. Méd. Montpellier,' 1881, p. 423.

[2] Thesis No. 23, 1882.

[3] 'Gazette de Montpellier,' 1883.

We shall discuss, in a later paragraph, this theory and several others propounded upon this subject. We must first produce our facts, and prove the relationship; the question of interpretation will come later on.

Our cases will fall into two groups. In the first, hysteria is the only manifestation of the tubercular diathesis. In the second, we shall find simultaneous pulmonary and hysterical manifestations. The two categories will serve to establish etiological relationship between the diathesis and the neurosis. The second alone will allow us to study the reciprocal influence which the two orders of diathesic manifestations appear to exercise upon each other.

I. OBSERVATIONS OF HYSTERIA, WITH TUBERCULAR HEREDITY WITHOUT PULMONARY MANIFESTATIONS.

CASE I. (personal).—*Neuropathic and Diathesic Heredity (Arthritism, and especially Scrofulo-herpetism). Hystero-Epilepsy, beginning at three years of age, with Scrofulo-herpetic Manifestations.*

J. C., a little girl of three years old, was seen by me with Dr. Bouffier, of Cette, who kindly furnished me with the notes of the case.

Neuropathic heredity.—Sister died at 8 or 9 months from meningitis, with violent convulsive attacks; brother died from cerebral accidents, during a laborious dentition, at 13–14 months. Maternal grandmother had severe attacks (epileptic?) and died insane. Mother very nervous. Maternal aunt has had violent attacks after strong emotion (fear of drowning).

Diathesic heredity.—Maternal grandfather has had cancroid of the upper lip. Mother very lymphatic: ganglionic enlargements, with attacks of eczema behind the ears. Maternal uncle: marked scrofulo-herpetic accidents. The sisters and brothers of the mother, says Dr. Bouffier, are all scrofulous, lymphatic, or of slow intellect. They are all below the average mentally, but those whose constitution is the best are the least developed in intelligence." Father lymphatic. Paternal grandmother rheumatic. Paternal grandfather has had rheumatic sciatica of several years' standing.

History of patient.—Carefully followed from birth, owing to cerebral affections of brother and sister, and has often taken bromide against the neuropathic heredity. Has had sometimes lymphatic enlargements of neck, and has had a kind of "mucous fever" lasting three or four weeks.

Last summer she had a great fright from falling into a tub, head foremost, after which she had an eczematous eruption on the face. She got better, but another emotional shock brought about another eruption, but less extensive. Three months after, during a walk with her father, was seized with a vertigo; and another followed two or three days after at the same hour, and under the same conditions. The same phenomena went on repeating themselves for a few weeks every two or three days. The attacks were very short, when, two months after the beginning, they were followed with loss of consciousness and resolution of limbs, and preceded by an aura, with alternative pallor and redness of the face, retraction of thumbs and convulsion of eye-balls. The attacks became more frequent. When I saw her she lost consciousness every afternoon at 4 or 5 o'clock, remained motionless, and as dead for about half an hour. No convulsions. She usually feels the attack coming on, gets into her mother's lap, and moans sometimes for a while. Or she is seized suddenly during her play or her tea. She often resumes, on coming round, her occupations where she had left them. Capricious temper.

I prescribe a continuation of the bromide, general cold ablutions, iodide of potassium and arsenite of soda, alternating with gold and sodium chloride, a series of blisters to the arms, (cod-liver oil is not well borne), and I mention a cure at Uriage for the following summer.

On October 28th, 1882, Dr. Bouffier writes: "The child seems much better. The summer passed without attacks, a few vertiginous attacks like the first were noticed at the beginning of the autumn, but have disappeared since. The temper is better." During the last two springs, she has had crops of small boils on the neck, face, and legs. This autumn she presents disseminated pemphigus bullæ and enlarged cervical glands.

In this case the hysteria is not doubtful, and the scrofulous diathesis is very clear in the heredity, and even in the aspect of the subject. Further, the neurosis develops itself without any of the great moral or physical perturbations which mostly bring about hysteria. The age of the patient and the surroundings in which she lives make, it seems to me, the pathogenic part played by the diathesis more clear and important.

CASE II. (personal).—*Scrofulous Diathesis.*—"*Hystérie fruste*" (*incomplete hysteria*) *of a chiefly sudoral nature.*

On Oct. 13th, 1882, a girl of 14 was brought to me, who has menstruated since the age of 11½ years, and who for the last three days presents extraordinary local sweatings. All the external clothing is soaked round the waist. She has saturated up to twelve napkins in a day; and actually (3 o'clock P. M.) wears the third flannel of the day. She feels at times, and very suddenly, a very violent pain, which constricts the lower part of the thorax, and immediately afterwards she feels herself inundated by an enormous flux of perspiration at the stomach; the liquid flows to the ground. These sudoral attacks have begun three days ago after a violent emotion, having been beaten by her aunt, who frequently ill-treats her. Menses have just appeared.

For the last eighteen months she has suffered from frequent and very loud abdominal noises, and for which she had already consulted me; extraordinarily intense borborygmi, during whole hours, simulating every kind of external sound, and very objectionable to the neighbours. At school she has also had frequent syncopes.

Her father is said to have died from abdominal cancer. The mother, whose conduct was bad, died of consumption when the child was 4 years old.

She has had numerous scrofulous accidents during childhood; disorders of the ears, protracted suppurations, chilblains, impetigo of the scalp, ganglionic enlargements, &c. She still has a deeply scrofulous aspect. Her body is weakly and spare. No thoracic symptoms, except a very slight dulness towards one of the external clavicular regions.

Atropia pills cut short the sudoral crises; after which I institute a chronic treatment with cod-liver oil, iodide of potassium, quinine, and blisters.

The curious (secretory) form of the hysterical manifestations would deserve some remarks if they were not beside our actual object. I shall only insist here upon the fact of the neurosis, together with the scrofulo-tubercular heredity and constitution.

CASE III. (personal).—*Neuropathic and Scrofulo-tubercular Heredity; Scrofulous accidents; Hysteria of a chiefly bulbar form.*

Miss X., aged 27, seen the 27th of June with Dr. Bompaire, who has kindly sent notes to complete the present observation.

Neuropathic heredity.—Paternal grandmother insane; father strange. Paternal uncle "has done foolish things which, without the intervention of the family, would have necessitated his reception into an asylum."

Diathesic heredity.—Mother suffers from marked herpetism. Three sisters; one died young after whooping-cough; another died at 11 years old, from "cerebro-spinal meningitis of rapid evolution (five days);" the third had scrofulous accidents at the time of puberty; abscesses in the inguinal and sternomastoid region: died tubercular at 18 years of age.

History of patient.—Scrofulous aspect; nose eczematous. Menstruation is regular, but began late. Frequent facial erysipelas (six or seven every year) between 18 and 23 years. At about 25, fixed pain about the epiphyses of the sixth and seventh cervical vertebræ; and apparition of fits occurring during the day, without loss of consciousness, beginning with coldness and numbness of extremities, with acceleration of pulse, and elevation of temperature, dilatation of pupils, redness of face, cutaneous hyperæsthesia, great volubility of speech, and convulsive jerks violently shaking the limbs and trunk. These fits occurred chiefly before, during, or after the menstrual periods, which have always been very regular. In the interval of the fits, great nervous mobility, and impressionability. At other times globus, nervous cough, &c.

At the moment of my visit all the phenomena are concentrated in the labio-glosso-laryngeal district; impossibility of swallowing other things than liquids. Some time ago she could not blow out a candle. At times, under the influence of an emotion, or without known cause, arrives the fit, which often begins with coldness of extremities, and is manifested by contraction of lips, cheeks, tongue and pharynx. Some time ago she could not put out her tongue, nor especially keep it out of the mouth. Water applied to the neck excites the fit; the patient cannot wash her neck.

I prescribed, besides the treatment against the hysteria itself, cod-liver oil, chloride of gold and sodium, and the application of a blister or the cautery. This treatment had no great result. "Since your visit," writes Dr. Bompaire, "the fits gradually diminished, then disappeared; then supervened nervous disorders of the pharynx, preventing the patient from swallowing solid or half-liquid substances. A metalloscopic examination seemed to show that zinc plates applied in front of the neck increased the symptoms, whilst gold seem to mitigate them. Several bottles of the auro-sodio chloride have been taken without alleviation, and the patient was sent (end of July, 1881), into the country at an altitude of 900 metres. There she could not go out once. She was seized with a 'nervous fever,' lasting three weeks, a remittent fever during which the pulse oscillated between 110 and 140, and the temperature between 38° and 40° C., with nightly exacerbations. The treatment consisted in the administration of quinine and aconite.

"During the last winter and spring the troubles of deglutition were complicated with an abundant sialorrhœa, the patient using as many as twenty pocket-handkerchiefs in the 24 hours. The saliva came chiefly from the parotid glands. At one time the weakness was so great on account of the deficient feeding, that peptones had to be employed, with good results.

"Since then the family decided to submit her to a quack treatment; and as I refused to conduct it myself, ceased to call me in. At the present time the patient's condition is more satisfactory. She can get up, and take a few drives. The difficulty of swallowing still persists."

Here again we find the double heredity, neuropathic and scrofulo-tubercular. Let us note in the subject the strumous manifestations and the remarkable (bulbar) form of the hysteria.

CASE IV. (personal).—*Tubercular Heredity. Rough breathing at the Apices, and frequent Bronchitic attacks. Hysterical fits, Rachialgia, &c.*

Mrs. G., aged 38.—Mother died after parturition, but slowly from consumption. Brother died, from slow consumption also, after the fatigues of the war.

History of patient.—First hysterical fits at 20 years, without apparent cause; grave, sometimes cataleptic; occurred repeatedly during several months. Regular menstruation. Three pregnancies; normal childbirth.

Six years ago she lost her husband, and left her home to look after an old, ill-tempered uncle. Protracted moral sufferings. Attacks occur again during eighteen months or two years, at variable intervals. Cauterisation of the cervix. Grave typhoid fever two years ago, with cerebral symptoms. Good health afterwards. In January 1881, during the financial crisis, she hears suddenly that the fortune of her children has been lost through secret speculations of their trustee; despair. Starts for Avignon, runs about the whole day, and in coming back in the train has a violent hysterical fit. This is the beginning of the actual disease. For several days fever and delirium. Attacks, at first frequent, disappear later on. Then, three or four months afterwards, great attack (with catalepsy). From the beginning until now, violent pains down the rachis, occasionally going through the thorax, preventing the dorsal decubitus. At the same time progressive onslaught of a paraparesis with bilateral anæsthesia. No urinary nor rectal symptoms, but at various times nervous vomiting. Cannot walk without a stick, does not feel the ground well. Cannot work, owing to pain caused by moving the arms.

On examination (October 1882), violent rachialgia. Impossible to press on the spinous apophyses. Sensations everywhere dull, especially on the left side, on the right breast, and as high as the knee in the right leg, and the elbow in the right arm. Nothing in the chest except some roughness at the apices. Coughs much since a fortnight. Frequent bronchites. Constant cold feeling in the lower extremity; the attack begins with the accentuation and the generalisation of this cold.

I send her to Dr. Regimbeau for applications of static electricity. The first day the very sight of the apparatus produces a violent fit. Afterwards the electricity is well supported. She leaves on the 1st of December, much better. I prescribe a lengthened treatment with the ferro-sodic arseniate.

Here there is no neuropathic heredity observed. But numerous and violent moral excitations have served as occasions to the tubercular diathesis to determine the neurosis.

CASE V. (personal).—*Tubercular Heredity. Hystero-Epilepsy.*

Mrs. B., age 32, was sent to me in July 1882, by Dr. Cayla, who kindly supplied some notes to complete the history of this patient.

Heredity.—Father died young of pneumonia. Mother died four years ago of heart-disease. The patient is the eldest of three daughters. One of her two sisters died phthisical at the age of 18, and the other, aged 21, and recently married, is hysterical. Mrs. B. has had three children. The one suffers from a general eczema, resisting every treatment; another died at birth.

History of the patient.—Lymphatic temperament; health good till first child was born. Fourteen years ago had stillborn child, upon which had grave eclamptic symptoms for about twelve hours; after then frequent sick headaches. The violent frontal pains become real seizures, occasionally accompanied with very short loss of consciousness, preceded with vertigo, and followed with loss of memory during a few seconds. No hysteria, globus, nor convulsive phenomena.

Little by little this condition became worse. The attacks become more frequent; they were characterised by a kind of aura, followed with vertigo and loss of consciousness. Feeling returned after a few minutes, but there remained for a few hours a complete forgetfulness of what had happened, some cephalalgia and muscular pains. Under the influence of fatigue, and menstruation, which had become irregular and insufficient, but few days intervened between the fits. Loss of flesh.

"After the second childbirth, two years ago, and an illness of her daughter, the nervous symptoms increased in intensity. She was seized with vertigo, placed the baby on the ground, without consciousness of that she was doing, went into another room, came back, and awoke astonished to find herself there. In the night she has left her bed, deposited the baby at the top of the stairs, and returned to her home. I saw her one day seized with a fit, of which she announced the invasion; she became slightly pale, contracted her hands and forearms. Then, with closed eyes, she walked to the middle of the room quite naturally, began to undress. Taken back to her arm-chair she came to herself, much astonished to find on her knees the petticoat she was wearing a few minutes before. In a short time she resumed her normal state."

She is weak, anæmic, of a very versatile temper; memory bad, affective feelings dulled.

I prescribed a hydrotherapic treatment at Lafoux, and on her return, a continuation of the cold-water treatment, progressive doses of arseniate of soda in glycerine, with bromide of sodium; and a purgative dose every month or so.

Dr. Cayla wrote to me in October 1882: "She went to Lafoux for a season, when the number and duration of the attacks were temporarily diminished. But on her return home she has not followed the prescribed treatment, notwithstanding the instances of her friends, and her state is much the same. I wished to send her again to Lafoux, but she has not consented."

Though tuberculosis has not been found among the ascendants, it seems to exist in the family, on account of the case of the sister. The facts are then so: of three sisters, one is phthisical, the two others hysterical, and the eczema in the daughter of one of the latter reproduces the classical manifestations of the scrofulo-tubercular manifestations.

Case VI. (*Second case of Paris, 'On the Hysteria of Little Girls.'* 'Thesis,' Paris, 1883.)—*Tubercular Heredity. Hysteria beginning at* 10 *years old.*

Heredity.—Mother died phthisical. Father in good health; ten brothers and sisters in good health also; two died of disease unknown to the grandmother who brings the girl.

Nothing noteworthy among the personal antecedents.

History of disease.—Nervous attacks at 10 years of age, then fits of somnambulism. Various painful symptoms; right ovarian pain, frequent sick headaches, anæsthesia of conjunctival and velum palati. Right hemianæsthesia.

We shall call attention to the presence here of a tubercular heredity in the absence of any neuropathic heredity and of any nervous over-excitation in the patient herself. Moreover, hysteria began early, at 10 years, a fact which connects the case with the first, and with the following.

CASE VII. (*Third case of Paris; loc. cit.*)—*Tubercular Heredity. Hysteria breaking out at* 9 *years of age.*

Heredity.—Mother died phthisical nine years ago.

Personal antecedents.—Ill-treated by step-mother; whooping-cough, measles, and scarlatina.

History of disease.—Nervous attack at 9 years of age, provoked by contrarieties and fears. Syncopes, with forebodings; ovarian pain on the left. Anæsthesia of conjunctivæ and soft palate; other symptoms.

Here again the tubercular heredity and hysteria are distinct, whilst neuropathic heredity does not exist. There has been, however, much ill-treatment, which may have acted as exciting cause to the neurosis. The latter became manifest at a very early age.

Similar phenomena are noted in the next six cases.

CASE VIII. (*Case* 12 *of Briquet*, p. 61.)—*Tubercular Heredity. Hysteria beginning in early youth.*

V. D. lost her brother from phthisis; has two sisters always ailing. She has been ill-treated by her parents, and from childhood till 29 years she had every nine or ten days a convulsive attack with loss of consciousness, and with all the characters of hysterical attacks. Worry had much to do with the production of these fits. The latter have ceased since, but there has remained until now (37 years), a well-marked general hysterical state. Two pregnancies between the ages of 25 and 30 have not brought about any fresh attacks.

CASE IX. (*Case* 13 *of Briquet*, p. 62.)—*Tubercular Heredity. Hysteria from childhood.*

A. C. has a hysterical mother. Father died phthisical, and one of her sisters died of convulsions. From her earliest childhood she has, when crossed, choking and spasm of the throat. At 7 years, complete hysterical attack; at 16, chorea; at 17, became again subject to hysterical attacks, which persist up to this time.

CASE X. (*Case* 14 *of Briquet*, p. 63.)—*Tubercular Heredity. Hysteria beginning at* 5 *years of age.*

V. P. lost her mother and sister from consumption; has had her first convulsive attack at 5 years of age, with loss of consciousness and after violent anger. This fit has all the characteristics of hysteria. Fits still persist now, at 24 years of age.

CASE XI. (*Case* 20 *of Briquet.*)—*Double Tubercular Heredity. Hysteria beginning in the* 7*th year.*

J. D. lost both father and mother from phthisis. Placed suddenly in presence of a dead body when 7 years of age she had a great fright, and was seized on the spot by a convulsive attack, with loss of consciousness. The succeeding attacks occurred at first several times a day, but gradually became less frequent, and disappeared at about 12 years, when menstruation began. Originally these used to be spontaneous, but later occurred only under the influence of some emotion. These seizures differed somewhat from ordinary fits; they were preceded by giddiness and tinnitus; then after a few minutes there was loss of consciousness, and when the patient recovered there was a choking sensation and epigastric constriction, but no crying. Still, notwithstanding this slight anomaly, the general character of the phenomena was too much like hysteria to leave the least doubt. Moreover, she has had in the hospital, ten years afterwards, when 22 years old, a well-marked and complete attack of hysteria, occasioned by a new fright.

CASE XII. (*Case* 20 *of Briquet.*)—*Double Tubercular Heredity. Hysteria beginning at* 9 *years of age.*

M. D. lost her father and mother, who died very young, from phthisis. When 9 years old she fell into the hands of relations, where her life, hitherto very quiet, became painful. She was ill-treated, and crying constantly, as if suffocated. She soon became subject to hysterical attacks, characteristic and complete; they were at first frequent, occurring several times a week, and caused by the least contrariety. They are now less frequent (17 years old), but she presents general phenomena of pronounced hysteria.

CASE XIII. (*Case* 31 *of Briquet.*)—*Tubercular Heredity. Hysteria beginning at* 8 *years of age.*

J. C. lost her mother, a brother, and a sister of phthisis. She has always been ill-treated; one day when 8 years old, after having been beaten more severely than usual, she had a complete hysterical attack, and from that time until 10 years of age she had similar seizures almost daily. They then diminished in frequency to increase again at about 15 years, when menstruation set in with some difficulty. Actually the girl, aged 29, has her attacks every week.

CASE XIV. (*Case* 35 *of Briquet.*) — *Tubercular and Neuropathic Heredity. Hysteria since childhood.*

J. D. has a slightly hysterical mother. Father died phthisical. She always was delicate, very impressionable, and much ill-treated by her parents. From childhood she suffers from choking and epigastric oppression every time she is scolded. Since then her health has always been indifferent. At 12 years of age she menstruated. Since about 15 or 16 she became subject to numbness of the limbs and muscular trembling. At 22 she began to have hysterical fits, and at 29 she is still in a general hysteric condition.

All these facts relating to hysteria beginning in childhood seem to me peculiarly important to our argument. The part played by heredity is here much more considerable, and that of external causes much less marked.

We can thus interpret in an especial manner the general numbers of the great statistics of Briquet. On 351 hysterical patients, the health of whose parents, brothers and sisters, has been ascertained with sufficient accuracy, we find: out of 282 fathers, 9 cases of phthisis and 9 of hysteria; of 327 mothers, 13 cases of phthisis and 103 of hysteria; of 338 sisters, 8 cases of phthisis; of 148 brothers, 10 cases of phthisis and scrofula.

No conclusions can be drawn from the crude numbers; but let us establish categories, and take into account the age at which the neurosis was developed. We then have 80 hysterical subjects before 12 years of age, that is, puberty; on 56 fathers, there are 6 phthisical or cancerous; on 78 mothers, 11 phthisical or cancerous (and 28 hysterical); on 81 sisters, 6 phthisical; on 43 brothers, 3 phthisical.

These numbers give per cent.:—

For hysteria in the adult, 3·19 % phthisical fathers; 3·79 % phthisical mothers; and 3·61 % phthisical relatives.

For hysteria in the child, 10·71% phthisical fathers; 14·86 % phthisical mothers; 13·07 % phthisical relations.

Having attracted the reader's attention to these facts, we resume our series of condensed cases.

CASE XV. (*Case* 66 *of Briquet*).

L. M., 22 years. Mother died phthisical; sister similarly affected. Delicate and very impressionable till 17 years, when first menstruation occurred; then suppression of menses. Left pleurisy with effusion (5 to 6 months), marked anæmia afterwards. Between 18 and 20 good health, menses regular. At 20, beginning of hysteria, due to great sorrow owing to death of sister. Various pains, convulsive attacks, rhythmical chorea, hyperæsthesia, and a series of obstinate symptoms leaving no doubt as to the diagnosis.

CASE XVI. (Lombard, 'Gazette Médicale,' 1876, p. 187.)—*Tubercular Heredity, but no Neuropathy. Hysteria beginning at the* 10*th year.*

Heredity.—"This case is one of a boy of 15, whose family I knew many years since, and where there is no epileptic antecedent. The father had good general health, and died from confluent smallpox. The mother is strong, with a good constitution, though somewhat lymphathic. An elder sister has always been well, excepting suppurated cervical glands. Another sister has had frequent erysipelas of the face during many years, and finally a pulmonary consumption, from which she died. Finally, a brother showed the symptoms of tubercular meningitis, which carried him off. Thus a family completely free from nervous disease, but subject to lymphatism and tubercles."

History.—At 10 years of age, fifteen days after a fall on the head, first attack of the evil. Five convulsive seizures between 1871 and 1875; globus hystericus, &c.; hemianæsthesia, and hemihypothermia on the left side; pain to pressure in the right iliac fossa.

The tubercular absence of neuropathic and heredity are well defined by the writer. A fall on the head seems to have been the existing cause of the localisation. Here again the family history is instructive: out of four children, one has suppurated glands, another a pulmonary phthisis, a third a tubercular meningitis, the fourth is hysterical. Why refuse to the neurosis its relationship to the other manifestations of the tubercular diathesis?

CASE XVII. (*Case 4 of Dunant.* Thèse, Paris, 1863.)—*Tubercular Heredity. Hystero-Epilepsy, and signs of Pulmonary Tuberculation.*

Heredity.—Father died consumptive; mother healthy; out of eight brothers and sisters three died phthisical, the others are well.

History.—Weakly constitution with signs of pulmonary tubercles (no other details). Patient appears to have been epileptic before the age of 20. During eleven months the author observes nothing but hysterical fits. Later, he witnesses an epileptic attack. Observation of a series of hystero-epileptic seizures.

The fact of the presence of pulmonary tubercles in this patient might have made us place this observation in the second category; but the absence of details concerning these phenomena made us prefer to include it here.

CASE XVIII. (condensed). (Observ. III. de Chambard, 'Du somnambulisme,' &c., 1861, page 85.)—*Tubercular Heredity. "Grande Hystérie."*

Heredity.—Father became alcoholic and dissolute after the death of his wife, and had vomiting of blood every month. Drunkenness was very prevalent amongst paternal ancestors. "Her mother died of chest disease, at 36 years old. She did not suffer from nervous diseases, no fits, her character was amiable but weak. The grandmother and two maternal aunts have also succumbed to pulmonary phthisis. Marie H. has two sisters: one died 10 days old; the other, aged 29, is alive, but in weak health, very nervous, and liable to frequent outbursts of anger, complaining of various ailments, of continual cardialgia and of leucorrhœa, which may be referred to a chlorotic and nervous state. She has two children, one a feeble-looking boy and an idiot girl. Her husband is entirely alcoholic."

Previous illness.—She had measles at 5 years old; then during the following five years she presented symptoms of scrofula, which were treated for a long time by cod-liver oil, and she also had a skin eruption. Menstruation occurred at 18 years; periods irregular, with lumbar and abdominal pains, nausea and vomiting. —No self-abuse, probably not even any sexual relation; no bad treatment, no privation.

Present illness.—At 21 or 22, had a nervous attack after some provocation, followed by different ailments in consequence, such as chorea during two days, repeated malaise, incessant vomiting with gastralgia, extreme mental uncertainty, passionate desire to attract attention, to please others, &c. Susceptible to mesmerism, she presents the different phenomena of somnambulism. Later on, rhythmical chorea (developed by imitation), with acute intellectual excitation, ending in true outbursts of mania.

The absence of neurotic heredity nervous overwork is remarkable, compared with the completeness of the tubercular heredity and the hysterical neurosis.

CASE XIX. (condensed). (Obs. XII. of Richer, 'Etudes Clin. ou Hystéro-Epil.,' p. 206.)—*Tubercular and Neurotic Heredity. Grande Hystérie.*

Madlle. X. 25 years of age. Mother and grandmother both died at 30 of tubercle. Father died mad in an asylum, after heavy domestic trials.

The first attack of hysteria came on at 19 years of age. Series of severe attacks of the type of hysteria major occurred.

CASE XX. (condensed.) (Obs. of Moreau de Tours. Cited by Richer, *loc. cit.* p. 352).—*Tubercular Heredity. Hysteria, attacks of Delirium.*

T., aged 42, shoe stitcher. Father died suddenly at 82. Mother died of chest disease, had been subject to nervous attacks.

No severe illnesses in her youth. Menstruation at 12 years of age, without attacks. Married at 24; had two children, who enjoyed the best health. The attacks came on after being abandoned by her husband; and in the form of delirium.

CASE XXI. (condensed). (Obs. I. of the 'Iconogr. photogr. de la Salpêtrière,' vol. iii.)—*Tubercular and Neurotic Heredity. Scrofulous in Infancy. Previous Hysteria.*

W., married, laundress, entered the service of Prof. Charcot at 18, May 16th, 1877. Father insane. Mother, laundress, died four years ago of chronic affection of the chest (cough, hæmoptysis); sober, nervous attack after annoyance only; was left an orphan at an early age. Nine children, of which five dead, four of convulsions, one of epilepsy (?) at age of 12. Of the survivors, the patient is the eldest; one boy of 18 has no nervous attacks, but has had hæmoptysis; another boy has been lost sight of; lastly, one girl seems subject to attacks of weakness.

Infancy of the patient.—At 22 months, convulsions, in consequence of which she became paralysed, deaf and dumb. Towards age of 7, condition improved. During this period she had outbursts of passion, measles, eczema of the head; chronic ophthalmia, which has left no traces; severe chilblains.

From infancy, fantastic disposition, attacks which became more marked at 12. Syncopal attacks; then in different forms, hemianæsthesia, &c.

CASE XXII. (condensed). (S. H. Serre. Cited by Mossé, 'Gaz. Hebdom. Sc. Méd. de Montpellier,' 1883.)—*Tubercular Heredity. Local Hysteria.*

Mother lymphatic. Hereditary antecedents, tubercular, on the paternal side. Age 19. Hysterical contracture of traumatic origin. Disappears suddenly, then reappears. Transitory aphasia. Various nervous ailments.

CASE XXIII. (personal).—*Scrofulous Diathesis* (Hereditary and personal). *Grande Hystérie developed in a Child.*

December 5th, 1882.—Little girl of 10 from Bize, sent to me by her medical attendant, Dr. Agostini, to whose kindness I am indebted for most of the following details.

Antecedents.—Mother nervous, having had an impetigo of the scalp during three years; at the age of 7, after vaccination, had for some time enlarged glands, and frequent eruptions on the nose. The maternal uncle presents the same features of scrofulous diathesis.

Present history.—Began with a kind of chorea; some disordered movements of small amplitude limited to the right upper limb; some slight convulsions in the muscles of the face. At times hiccups lasting sometimes for hours, accompanied by shrieking, which could be heard in the street. After unsuccessful use of various remedies (assafœtida, chloroform, chloral, compression of the phrenic), sulphate of quinine made the attacks disappear; however, they reappeared after some weeks' respite, but were always subdued by the sulphate of quinine. The spasm ceased five or six months before my first examination, but then occurred suddenly a partial paralysis of the right lower limb. The child could no longer walk without the aid of a stick, and complained of pain in the corresponding knee, which was not, however, either red, hot, or swollen. During that time the choreic movements had nearly disappeared. After two months' duration the paralysis gradually decreased, and the day it disappeared completely the choreic movements returned with increased severity. "The alternation between the paralytic and the convulsive phenomena was not altogether complete, but almost so." Some time after, fresh convulsions appeared of the same kind as the first, but limited this time to the stomach; fruitless efforts of vomiting, like a person vomiting with an empty stomach, and this five or six times a day, sometimes more. The appetite, capricious in the intervals between the attacks, then becomes very keen, a kind of boulimia. The sulphate of quinine suppressed these phenomena, which reappeared a month afterwards; the same remedy again succeeded; but then failed the third or fourth time. The hydro-bromate of quinine then succeeded, to fail again. Rivière's mixture also succeeded at times—she was treated at one time simultaneously with iodide and bromide of potassium, and cold lotions, douches to the vertebral column, cinchona, arseniate of soda, iron, and sulphur baths.

After examination, I diagnosed hysteria of a scrofulous nature (predominance of the choreic phenomena) and I prescribed—sulphur and arsenical baths in turn (later on, wet packing, chloride of gold and sodium, and extract of valerian), a belladonna plaster at the pit of the stomach, and the cautery or repeated blisters on the arm. In the spring she was to be sent to a hydropathic establishment or to a mineral bath.

On her return to Montpellier, and before beginning any treatment, the spasms of the stomach ceased, and were replaced by attacks which came on in the night nearly always at the same time, towards 4 or 5 in the morning, consisting of a profound sleep from which the child is awakened with difficulty, and during which she snores considerably. If awakened, she rises up quite astonished, and the snoring ceases, but she complains of headache and giddiness. This giddiness returns even during the day. If left in bed, she is immediately seized with an irresistible desire to sleep, and the snoring attacks return at once. During this time she yawns very often, sneezes, and only awakens after having been shaken. The valerianate of quinine given by Dr. Agostini, influences the attacks and retards them.

These new phenomena confirm me in my diagnosis; I keep fundamentally to the same treatment, advising the valerianate of ammonia and the magnet of Charcot.

The 9th of May, 1883, Dr. Agostini wrote to me :—"At the same time I wish to ask you to which watering-place it would be best to send the little girl suffering from hysterical chorea that you saw this winter, and who is going on pretty well on the treatment prescribed. Since you saw her she gesticulates less, but the gastric crises continue in spite of everything; first she has hiccups for two or three days, then nausea and vomiting, then the sleeping attacks, then she again is free from anything; but she never remains a whole week without having something. It is a true visceral chorea."

On the 25th of June the child was brought to me. A fortnight before, a scrofulous kerato-conjunctivitis showed itself in the left eye; since then all the other attacks disappeared. I prescribed a course at Lafoux (hydropathic establishment), then a course at Uriage (sulphur and sodic chloride waters). From this latter place Dr. Teulon Valio (to whose care I had entrusted the patient) wrote to me on the 23rd of July: "The hydro-mineral treatment has been adopted with a view to influence the scrofulous diathesis and to modify the symptomatic manifestations: baths of gradually increased strength and duration, and alterative and purgative waters to drink. I should have kept to these purgative waters if the parents of the patient had not drawn my attention to the fact that the purgatives were always followed by a return of the nervous attacks. The four first baths passed off well. After the fourth, a sleeping crisis came on. To prevent their return, and relying on the comparatively satisfactory results obtained at Lafoux by moderate douches, I prescribed a very weak Scotch douche.

"This treatment not having produced the expected result, I had recourse to baths which act with more energy on the entire system. Since yesterday the sleeping attacks have not returned; but have been replaced by a fit of hiccup. This attack was far less violent than the preceding ones, but was followed by a very marked weakness of the left inferior limb. The patient can only walk supported by her friends, and complains of a pain in the tibio-femoral joint. This pain is not increased either by pressure or by flexion or extension; walking only brings it on. There is not the least febrile reaction." The cure was continued very successfully, *everything* disappeared, and I found a great improvement in the patient when I saw her the following 8th of August.

The crises, however, reappeared in the form of hiccup, vomiting, sleeping or true somnambulism. With regard to this latter species of phenomena, Dr. Agostini writes on the 22nd of October:

"If the sleeping crises disappear for a few days, we are sure to see the return of the hiccups, or of the nausea with or without vomiting. These nervous sleeping crises generally happen in the morning, but can appear at any hour of the day, and last, as the case may be, half an hour, an hour, or more. Two days ago a sleeping crisis lasted seven hours, from which it was impossible to awaken her, in spite of all the means used. These attacks, which before were simple crises of nervous sleep, during which the child remained quietly in bed, are now crises of somnambulism. Although the child's eyes are shut, she sees and hears everything during the crises, knows who comes into the room by their step and walk. If anything unpleasant is said to her, or if they threaten to wet her, or make her smell something, she gets angry, and pushes everything violently away that is offered her. She replies by signs to all questions, and if she wants anything, asks for it by gesture: if she wants to drink, she puts her finger to her lips as if in the act of sucking, and if not understood, becomes irritated, gets up, and leads the father or mother to the sideboard or cupboard where the things she wants are, and always with the eyes shut. She is able to go through the whole house, I do not say quite alone, because they do not allow her to walk alone, but with help, and she directs the way wherever she wishes to go. During the crises she even sometimes amuses herself with a little dog, which she makes jump over a stick resting on the cross-bars of two chairs opposite each other, and according as she wishes the dog to jump higher or lower, she moves the stick to the lower or higher bars of the chairs; and that with eyes always shut spasmodically. During the whole attack she cannot talk; but towards the end she speaks, although not yet recovered from her state of somnambulism.

"When the crisis is over, the child remembers nothing of what she has said or done in her sleep, and is much surprised at everything told her."

I advised them to try artificial hypnotism, and above all a complete course of hydrotherapy and statical electricity, carried out, if possible, away from the patient's family.

This case is remarkable for the clear manifestation of the "grande hystérie,"—without convulsive attacks, however—on a basis manifestly of scrofulous heredity.

Case XXIV. (personal).—*Hysteria (Polyuria, incomplete Exophthalmic Goître).—Tuberculous Heredity.*

Madame G. was sent to me by Dr. Dargelos of Aix, September 27th, 1883, aged 42. Her mother died young of a long illness (of the liver it was said); two relations (uncle and aunt) died tuberculous. An uncle died of diabetes; another of heart-disease.

Menstruation at 10 years of age; for the last four or five months, periods have been at shorter intervals; delicate health; very impressionable; lively imagination. Ten years ago, after violent emotion, attacks of hysteria (unconsciousness, loss of words, and severe convulsions, "grands mouvements;" ending in weeping), every day for a month; they began at a monthly period (accidental suppression), and ceased at the following period. Frequent cephalalgia, almost constant, very violent occasionally. In August 1883 two new attacks; at this time a great diminution of strength, pruritus vulvæ intolerable, intense thirst and considerable polyuria, seven or eight times in the twenty-four hours; no sugar, no albumen; density 1004 to 1006. Further, at the time of my examination, very decided swelling of the thyroid body, particularly the left lobe; no exophthalmia; frequent and violent palpitation.

I prescribed hydrotherapy, bromide of potassium, iodine ointment, and tonics.

In spite of the absence of exophthalmia, the symptoms, we think, are those of (incomplete) Graves' disease, which is well known in relation to hysteria. Here it is one of the manifestations of severe neurosis, very marked; and on the other hand tuberculosis is present; only it is mixed with other hereditary taints, which makes the case less conclusive.

Case XXV. (personal).—*Tuberculous Heredity. Hysteria. (Neuropathic condition.)*

M. P. of Marseilles came to consult me on Nov. 1st, 1883, age 35.

Heredity.—Slightly neuropathic in his mother and maternal grandfather. Father and brother died tuberculous. He himself up to 5 or 6 years old was, as he said, "full of humours;" wets his bed at night up to 15 years. Masturbation; venereal excesses, no syphilis. Marries at 25 a woman, who died of chest disease a year ago. Four children; two died very young, one of the survivors weak.

His illness is essentially characterised by sensations of constriction of the throat (globus), which occurred after the onset of frequent seminal losses, and pain and weakness in the loins. General nervous condition very marked; incessant distraction; continuous work quite impossible; head weary; imagination constantly at work. No memory, &c.; has taken chiefly bromide of potassium, about 20 grammes a day.

I advised him to leave off the bromide, and ordered hydrotherapy, arsenic in glycerine, and the constant current along the spinal column.

I admit that here the hysteria is incomplete, many would call it simply neurasthenia, all these neuroses, however, belong to the same family. The tuberculous heredity is very marked.

Here we conclude the observations of our first group. The eight personal observations, and the seventeen borrowed from various authors (in a rapid review of the authorities), seem to me to prove that tuberculous or scrofulous heredity is more frequent in hysteria than is commonly believed. I mean hysteria of the usual type, and which has not any extraordinary development.

The family histories which we have described tend to show that hysteria may probably represent, in some members, the diathesis which the more typical phthisis shows in others.

Thus, from my point of view, the doctrine is exemplified in cases like observation V, where, of three sisters, one is phthisical, and the other two hysterical; the daughter of one of these latter having already the classical symptoms of the scrofulous or tuberculous diathesis. This case cannot be ascribed to atavism. The chain of the diathesis has not been broken; it only is represented in one generation by hysteria, instead of by some form of chronic pneumonia.

The family history of observation XVI is just as instructive. Of four children, one has suppurating glands; another, pulmonary phthisis; a third, tubercular meningitis; and the fourth, hysteria. Most medical men consulted for the first three would declare the unity of the scrofulous-tuberculous diathesis, and will make an exception for the fourth. There is no good reason why the same argument should not be used and the same scrofulo-tuberculous nature be asserted for the nervous hysteria itself.

We shall complete the exposition of the doctrine and its applications, after having given the observations of our second group. Up till now we have noted nothing in the thorax of our hysterical cases; already, however, we have seen in some of them scrofulous manifestations coinciding, or oftener alternating, with the nervous. In the following observations there exist in the same subject hysteria and pulmonary tubercle, so that one can study the relations between the two forms of manifestations of the common primary diathesis.

(*To be continued.*)

ON POSTERIOR SPINAL SCLEROSIS, CONSECUTIVE TO DISEASE OF BLOOD-VESSELS.

BY THOMAS BUZZARD, M.D.,

Physician to the National Hospital for the Paralysed and Epileptic.

(MICROSCOPICAL EXAMINATIONS OF THE CORD, BY W. BEVAN LEWIS.)

TIME does not appear to have done much towards clearing up the difficulty of deciding a question which really occupies the very threshold of enquiry into the etiology of tabes dorsalis. It is generally though not, I think, universally, agreed that in this disease we have to do with an irritative lesion of the spinal cord, not a simple atrophy. The question, however, may still be asked, as it was put by Charcot ten years ago:[1] "What is the primary seat of this irritative lesion? Is it the neuroglia? Is it, on the contrary, the nervous element?" He goes on, it will be remembered, to say: "I acknowledge that, considering how the change remains confined systematically, in a way, to the area of the posterior fasciculi, the borders of which it does not spread beyond unless in exceptional circumstances, I cannot help inclining strongly to the second hypothesis. The parenchymatous irritation would then be the initial fact; the interstitial irritation would be secondary."

The remark is made in connection with systematic, fasciculated sclerosis, such as is found in tabes dorsalis, and in primary sclerosis of the antero-lateral columns. As regards disseminated sclerosis, Charcot speaks more positively. In his judgment "the multiplication of nuclei, and the concomitant hyperplasm of the reticulated fibres of the neuroglia, undoubtedly constitute the initial, fundamental fact and necessary antecedent; the degenerative atrophy of the nerve elements is consecutive and secondary." The hyperplasia of the walls of the blood-vessels, he goes on to say, plays merely an accessory part. In the affection of the neuroglia he finds all the characteristics of "formative irritation." Disseminated sclerosis is a primary and multilocular chronic interstitial myelitis or encephalitis.

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[1] 'Leçons sur les maladies du système nerveux,' IIe serie, 1er fascicule, p. 6.

So that, according to this view, the relation in point of time is exactly reversed in the two conditions. In the one case (fasciculated sclerosis) it is the nervous element proper which first shows "irritative lesion;" in the other (disseminated or insular sclerosis), the connective tissue. In spite of this fundamental difference in the process, the results appear to be the same. As regards the histological characters, grey induration in locomotor ataxy, according to Charcot, differs in nothing essential from what it is in disseminated sclerosis, or in symmetrical sclerosis of the lateral columns.

Wilks and Moxon, I observe, differing in this respect from Charcot, draw a distinction between "sclerosis" and "grey degeneration,"[1] because they do not find the same microscopic appearances in the "insular" as in the "ribbon-like" form of sclerosis. In the "insular" form, especially in the brain, they meet with numerous granule masses which they have not generally found in the grey degeneration of posterior columns, "whilst this," they say "is softer." On the other hand, they agree with Charcot in believing that, whilst the change originates in the neuroglia in insular sclerosis, it begins in the nervous elements themselves in the grey induration of tabes. Bastian [2] remarks that "the patches of sclerosis which constitute the anatomical basis of insular or disseminated sclerosis do not differ in their essential nature, or in their appearance (macroscopic or microscopic), from the similar overgrowths of the neuroglia that occur in locomotor ataxy and primary lateral sclerosis." If I do not misunderstand him, Bastian believes that in locomotor ataxy, as in disseminated sclerosis, the change in the nerve elements proper follows the overgrowth of the neuroglia. As regards the process of sclerosis, he asks: "Does it begin in the connective tissue elements themselves? or is there some primary change in the small vessels (possibly of the nature of endarteritis) leading to obstructions and a sequential overgrowth of the neuroglia?"

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[1] 'Pathological Anatomy,' 2nd ed., 1875, p. 250.

[2] Quain's 'Dictionary of Medicine,' article "Spinal Cord, Special Diseases of," 1882.

In another place[1] I have described a case which in its developed form was not distinguishable by any peculiarities from an ordinary example of tabes dorsalis, yet had manifestly travelled to that condition through a phase of meningo-myelitis, and I suggested as probable that the inflammatory action might have attacked the connective tissue of the cord through the channel of the inflamed pia mater. The case which I am about to bring under observation is one which, I think, tends strongly to confirm the probability that such a process is, at all events, an occasional mode by which tabes dorsalis is produced. The case is an important one for several reasons, but principally because, owing to its fatal termination, it has admitted of complete examination, and I have had the great advantage of receiving from Dr. Bevan Lewis, who was kind enough to undertake the task, an admirably detailed account of the microscopical appearances in the cord, basal ganglia, and a portion of sciatic nerve. Mr. Nettleship also has been so good as to present me with a valuable report of the changes in the optic nerve, of which I am very glad to avail myself. Deferring for the moment further consideration of the general question, I will now proceed to describe the case.

Henry L——, æt. 39, unmarried, was admitted into the National Hospital for the Paralysed and Epileptic, under my care, on October 13th, 1881, when the following notes were taken by Dr. Beevor, then Resident Medical Officer.

The patient has been a house-painter for the last seventeen years. He has never suffered from lead-poisoning, colic, or wrist-drop, and there is no definite blue line on the gums. He has not been a hard drinker.

In his family history there is no paralysis or fits. His father's sister was insane. His mother died of consumption.

He has never had scarlatina or rheumatic fever; has twice had gonorrhœa, and once, four years ago, had a chancre, which was not, according to his account, followed by eruption on the skin, or sore throat.

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[1] 'Clinical Lectures on Diseases of the Nervous System,' 1882, p. 207.

He dates his present illness from twenty-two months ago, when he caught cold, from having been much exposed during the winter. His illness began with tottering and weakness of the legs, which used to give way with him, and throw him down. He found that he had especial difficulty in walking in the dark. At the same time he had numbness and loss of sensation in the legs, and also some numbness in the ends of the fingers. He has not been able to walk by himself for the last twelve months.

For the last eighteen months he has suffered from shooting-pains, especially in the knees and thick part of the thighs; less in the shins and ankles. He has never had shooting-pains in the head, trunk, or arms. For about a twelvemonth past he has experienced the feeling of a tight pair of stays compressing the lower part of his trunk, together with a heavy pressure all over his head.

His sight began to be affected two years ago, when, as he describes it, his eyes became dim. Two months ago he began to see things double, and then gradually he lost sight in the left eye. He had a blow on the left ear about four years ago, and lost his hearing on that side.

He has never had any attacks of vomiting or pain in the stomach. For the last twelve months he has had some difficulty in using his hands, and has not been able to write during this period. His urine, for the last six months, has been apt to run from him on any exertion, and he has not always been able to pass it on voluntary effort. He has not suffered from nocturnal emissions; his sexual desire has diminished.

Patient is to all appearance a strong healthy-looking man. His lower limbs are well developed, and are not at all wasted.

He cannot walk or even stand without help, and is unable, as he lies, to lift either foot off the bed, though in this posture he can flex his knees, and can also flex and extend the ankle-joints. In his judgment the left leg is stronger than the right. With assistance he can just manage to stand on his feet. The arms are not wasted, and are indeed well-developed. When his eyes are closed, he has great difficulty in touching the tip of his nose with either hand. The grasp of the right hand measures 25° by the dynamometer, that of the left, 50°.

The patellar tendon-reflex is absent on both sides; there is no ankle-clonus. Direct percussion of the vasti interni causes contraction of the muscle, which is well marked. Percussion of the tibialis anticus produces contraction more readily than normal.

The plantar reflex is obtained after a delay, which is equal on the two sides. The abdominal and epigastric reflexes are also obtained on each side. The cutaneous sensibility of the legs is much affected. He cannot feel tactile impressions from the knees downwards, but can localise correctly sharp pricks with a pin. There is, however, a delay of from four to five seconds in the perception of pin-pricks in both soles of the feet. In his arms he usually succeeds in localising tactile impressions. With the left eye he cannot tell light from darkness. With the right he can read No. 14 Nettleship with difficulty at two feet.

The left eye, by the ophthalmoscope, shows the characteristic signs of advanced atrophy; the right is unduly pale.

A watch is heard at a distance of three feet with the right ear, not at all with the left. A tuning-fork touching the teeth is heard in the right ear, not in the left.

The heart-sounds number ninety-six in the minute, and are indistinctly heard. There does not appear to be any murmur. The urine contains no albumen. The right pupil measures 4·5 mm., the left 5 mm. Neither reacts to light; each contracts during accommodation.

Under treatment with iodide of potassium some improvement was noted in this man's condition when he had been in the hospital a couple of months. The pains were described as not being so bad or so frequent, and there was a little more power in the control of the bladder. But towards the end of January he was attacked with tonsillitis, which was accompanied by prostration, from which he never rallied. Retching and vomiting, accompanied by "screwing" pain in the abdomen, began in the beginning of February, and continued with scarcely any intermission until his death. After the attack of tonsillitis, his legs became quite powerless—the urine dribbled away unconsciously, and he entirely lost control over the bowel. The sight of the right eye became worse. (He was quite blind with the left.) There was a tendency to formation of bed-sores in various parts of the lower extremities, but, with attention, this did not increase. For about a fortnight before his death the vomiting was constant and no food was kept down. The pains in the legs continued to attack him. Nutritive enemata were regularly administered, but were not retained. The greatest relief that he obtained (though this was but small) was from hypodermic injections of morphia. He died on March 6th, 1882.

According to his own account, the duration of this patient's illness from first to last was twenty-seven months. At the autopsy, which was made by Dr. Coxwell, the body was found somewhat emaciated. There were no bed-sores.

On removal of the vertebral spines and laminæ the dura mater was found to be distended by a considerable excess of sub-arachnoid fluid. The dura appeared somewhat hyperæmic, but this was possibly post mortem. No inflammatory exsudation was visible. On slitting open the dura mater from below upwards, numerous (about 20) small round calcareous bodies were remarked. These were about one or two centimetres in diameter, flaky in appearance; some circular, others more irregular in outline and they were attached to the parietal layer of the arachnoid. The soft membranes were thickened and adherent generally.

The posterior columns of the cord appeared grey to the eye. (The spinal cord and mesocephale were sent to Dr. Bevan Lewis for microscopical examination.) The brain appeared healthy. The lungs were œdematous, but crepitant, the heart healthy. As regards the kidneys, in each the capsule peeled readily, and the cortex appeared somewhat pale. On the surface of the right kidney there were numerous minute abscesses, the size of a hemp-seed and smaller; the infundibula were much dilated and the papillæ flattened. The ureter was greatly dilated, as also was the pelvis of the kidney, which was lined with greenish pus.[1]

In the left kidney the pelvis was dilated and injected. It contained much fluid pus. The papillæ were greatly flattened.

The walls of the bladder were much thickened, the increase being due to hypertrophy of muscular substance. There was no ulceration of the mucous membrane.

There was atrophy of the intrinsic muscles of the thumb in each hand.

REPORT OF MICROSCOPICAL EXAMINATION OF THE SPINAL CORD.

BY W. BEVAN LEWIS.

THE spinal cord is diseased throughout the whole length of its posterior columns, the dorsal region having suffered most profoundly, the lumbar region implicated to a minor degree, whilst the cervical portion is the least affected by the lesion. The dorsal cord is specially notable for its small size, its shrunken aspect, and a twisted or distorted contour of its upper half, due to disease of its central tracts; the cervical and lumbar regions are less perceptibly atrophied.

The appearance of the diseased columns in transverse sections of all regions of the cord is to naked-eye examination that of grey degeneration, the chrome coloration being much deeper in the healthy columns than in the diseased tracts, which latter become translucent in glycerine as contrasted with the opacity presented by the healthy medullated columns.

Sections could be obtained with ease from the cervical and lumbar regions, but the dorsal cord, especially in its upper half, was so degenerated and otherwise altered, that it was only with extreme difficulty that tolerably satisfactory sections could be secured.

With low power objectives all stained sections alike exhibit extensive sclerosis of the posterior columns, both the columns of Goll and Burdach being deeply involved, and the former throughout their whole vertical extent. As will be more minutely particularised in describing the regional distribution of the lesions, the degenerative process had extended from the posterior radicular zones as far as the posterior nerve-roots, and, sweeping across them, had invaded the direct cerebellar tract, and in many cases the crossed pyramidal tract.

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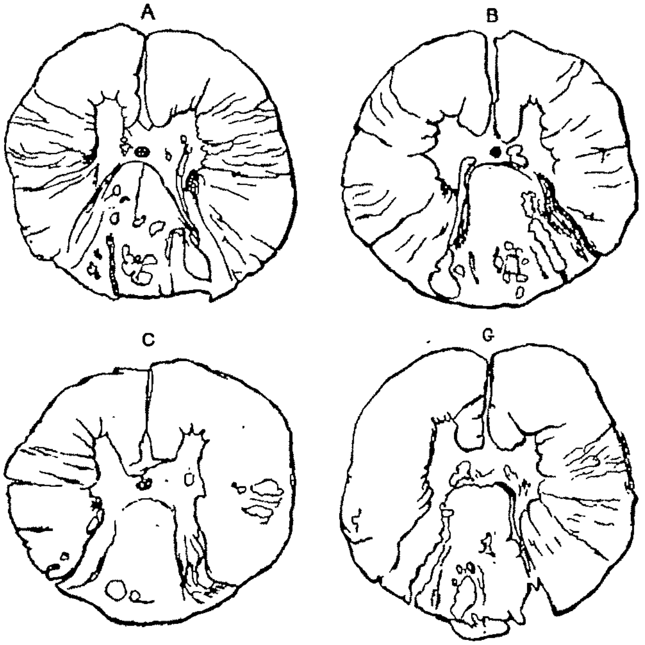
[1] The pyelitis was evidently secondary to the disease of the bladder.

In those regions stamped by the greatest intensity of morbid change the anterior cornua had invariably suffered, the various groups of cells being either greatly wasted or wholly absent, and the hemispheric distribution of the lesion was such that, whilst one horn might be entirely free from disease, the opposite cornu might be found shrunken to one half its proper dimensions.

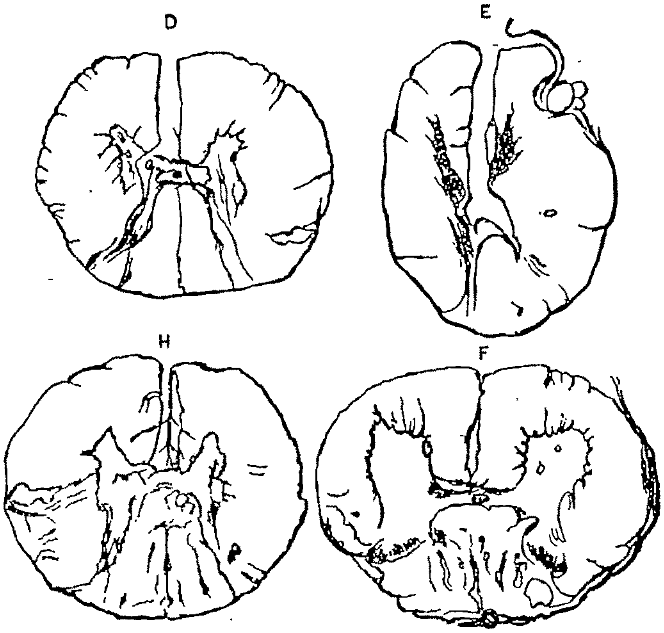


A, B, C, G. Channelling of posterior columns and cornua by diseased vessels in the dorsal region of the cord.
C. Implication of lateral columns in the dorsal region.

All the structures invaded by the degenerative process exhibit a deeper staining by aniline and other dyes than does the healthy tissue, whilst picric-acid preparations show the characteristic *translucent* aspect of sclerosed nerve tracts.

Even with low power objectives these areas exhibit an apparent remarkable degree of vascularity, the vessels being extremely numerous and dilated, their coats thickened and diseased. In fact, the wide-stretched orifices of these numerous vessels, with their peculiarly thickened walls, was a most notable feature, as they crowd the field in all directions, invariably forming the centre of a patch of fasciculated sclerosis.

In the *dorsal region* of the cord were first met peculiar *pin-hole cavities*, usually circular or oval in contour, with sharply defined margin, extending down along the course of the medullated strands, or horizontally disposed as elongated excavations or channels directed from the peripheral to the central aspect of these columns. Such cavities look as though *punched out* of the tissue, and on cutting down through the dorsal region they become more and more numerous, but almost invariably limited to the posterior columns. Occasionally a large cyst-like cavity would be met with, measuring 1·5 mm. to 2 mm. in diameter, or a linear series of such in moniliform arrangement would be seen running parallel to the posterior cornu of one hemisphere along its radicular aspect (Fig. G). Still lower in the dorsal cord such a cavity would be seen in the caput cornu, or at its base, on one occasion through the commissural connections of the anterior horn, and also in the



F. Channelling of posterior columns and cornua by diseased vessels in the lumbar region of the cord.
D, H. Implication of lateral columns in the dorsal region.
D. Lesions of commissure in the dorsal region, exhibiting also atrophy of anterior cornua.
E. Shrinking of the dorsal region of the cord (upper third).

direct cerebellar tract of one side of the cord (Fig. B, D.). The contents of the cavities were invariably lost in making transverse sections, and their nature was only satisfactorily determined by means of vertical sections through the columns. In such vertical sections diseased vessels were seen in large numbers passing upwards through the sclerosed tracts with many *ampullar* enlargements along their course—these enlargements occupying the spaces above referred to—which are therefore identified as unnaturally expanded perivascular channels. These nodose and moniliform enlargements along the vessels were not aneurismal, but were due to vast accumulations of large nucleated cells which occupied the perivascular sheath of the vessel. The actual calibre of the vessel was sometimes dilated; at other times it appeared constricted by the encircling mass of cells; whilst occasionally, although *rarely*, a genuine aneurismal condition of the vessel had been produced along its diseased tunics. The moniliform channels were occupied by a corresponding moniliform series of such cellular outgrowths upon the vascular walls, and thus it appeared that the whole dorsal region of the cord had been subjected to an invasion by an extensive peri-arterial affection, and its medullated strands exposed to destructive pressure from these nodular outgrowths on the vessels.

The investing membranes of the cord were notably thickened, closely adherent to the surface; their vessels tortuous, enlarged, and similarly implicated where they lie in proximity to the posterior columns. These diseased vessels from the pia mater pass into the substance of the cord through tracts of sclerosed nerve tissue, which lie on either side of them, and more or less definitely appear to follow their various ramifications. The vessels invariably exhibit thickening of their coats, proliferation of their cellular elements, and a peculiar vitreous lustre; whilst the cells crowding the perivascular lymph sheath have each one large nucleus, nearly as large as its investing cell, readily stained by carmine and aniline, and apparently derived directly from proliferation of the endothelial elements of the lymphatic sheath. The sheath throughout its whole course contains large numbers of such elements, but they chiefly congregate in nodular heaps around the vessel as above described.

It appears to me that the peri-arteritis, originating in the membranes and spreading inwards along the nutrient supply of the posterior columns, plays the chief rôle in the production of the lesions to which the medullated strands have succumbed.

As to this presumably *secondary* lesion induced in the nerve elements, the following points were noted.

In all vertical sections of the posterior columns are observed vast numbers of oval-shaped bodies, arranged more or less in linear series along the direction of the medullated fibres. These oval bodies are coarsely granular, and strongly stained of a deep yellow or yellowish-brown tint, and when treated by osmic acid, invariably show a *darkened nuclear-like body* in the midst of the granular elements. The medullated sheath is itself peculiarly affected by *large and irregularly distributed varicosities*—scarcely a fibre was detected unchanged in some part of its course in this manner. The axis cylinder does not appear enlarged, but often contorted, twisted out of position by the changes undergone in the medullated sheath, or obscured in part of its course, and often entirely unrecognised. These diseased medullated fibres are separated by abundance of fine fibrillated tissue, specially well seen in vertical sections.

The moniliform varicosities of the nerve fibres were due to segmentation of its medulla through proliferation of its interannular cells; these interannular cells being represented together with cellular elements around the fibre by the ovoid granular bodies containing deep-stained fragments of myeline (on osmic-acid treatment). In all respects the changes here observed were identical with those lesions seen, after section of a nerve-trunk, in its peripheral segment.

Such was the nature of the lesions found in every morbid portion of the cord, whether the white medullated columns or the grey matter was the subject of examination.

THE CERVICAL CORD.

a. Posterior Columns.—Viewed by low powers, the whole of these columns are found more deeply stained than other parts, the deepened tint continued backwards along the posterior horns across the roots of the spinal nerves, and beyond them to the direct cerebellar tract. An apparently healthy portion of tissue intervenes between the posterior horns and the diseased part of the radicular zones. The columns of Goll are invaded symmetrically on each side, the innermost portion, however, near the posterior commissure, showing comparative immunity from fibroid change and atrophy of nerve elements. The transverse sections are characterised by the notable inequality of the nerve elements. Greatly enlarged medullated tubuli are scattered here and there over the field, whilst intervening fibres are atrophied, or almost replaced by finely punctated fibrous tissue. The large swollen tubuli represent sections across the varicosity of a fibre—the smaller elements being sections across the more constricted parts of the fibre.

The more deeply stained diseased areas owe their intensity of colour not only to increased connective, but to the vast number of abnormally large and thickened vessels which form a notable feature both in the columns of Goll and Burdach. The internal and external posterior arteries (Ross), together with the arteries of the commissure, and the radicles for the posterior horns, have all suffered from the inflammatory changes in their lymphatic sheaths, and the internal posterior nutrient branch is especially implicated; it is enlarged, and takes a tortuous course as if elongated, through its sheath, which is stretched tense and smooth over the vast accumulations of nucleated cells crowding the walls of the vessels. In no case has hæmorrhage occurred into the sheath, or into the substance of the cord.

From near the level of the sixth cervical nerves downwards the destructive lesions are most severe, but the columns of Burdach are less involved at the level of the fifth cervical nerve, and from this point upwards the extent of morbid action is greatly diminished, so that at the level of the third nerve two narrow ribbons of sclerosed tissue alone are seen, one in each radicular zone, separated by a fairly wide tract of healthy tissue from posterior cornu and commissure.

The posterior roots of the spinal nerves, moreover, at this site issue from the cornua in clearly defined healthy bundles, whilst lower down the cervical cord these roots pass through sclerosed tissue extending into the cerebellar tracts.

β. Lateral Columns.—As before stated, the *direct cerebellar columns* are involved by extension of the lesion along the margin of the posterior columns outwards across the posterior roots. The entire area of these fibres is implicated in both hemispheres of the cord, the morbid change mapping them out most distinctly from the crossed pyramidal tract. The *crossed pyramidal* and other tracts of the lateral columns are in this region perfectly healthy.

γ. Anterior Columns and Root Zone.—The columns of Turck are healthy. The anterior root zone shows a morbid change, limited to one hemisphere, and most distinctly apparent at the level of the fifth or sixth cervical nerves. The hemisphere alluded to has a rounded contour in contrast to the broad sweep of the opposite and more healthy segment. This alteration is to be attributed to central changes in the corresponding cornu, as will be hereafter seen. The anterior roots in this altered segment, however, when contrasted with the opposite nerves under a low objective, are seen to be far less defined and sharp in the contour of the individual fibres. With higher powers they are seen to have very tortuous axis cylinders, which, although not swollen in any part of their course, are very indistinct, as though interrupted by the *varicose enlargements of the medulla* of the sheath, which are also apparent here. Occasionally, though rarely, a similar change is met with in the *descending medullated strands* of the anterior root-zone in this hemisphere, for here and there may be seen in these columns an enormous pear-shaped swelling of the medullated sheath, into which plunges an axis cylinder, to be lost from view in its interior, again traced further on at a subsequent constriction of the medullated sheath.

δ. *Anterior Cornua.*—An extension of the lesion was traced into the anterior horns at the level of the fourth and fifth cervical nerves, implicating one hemisphere much more seriously than the other.

The hemisphere thus involved is the one already referred to as rounded in contour, a condition undoubtedly due to shrinking subsequent upon scleroses and degenerative changes within the corresponding horn of grey matter, associated, as was shown, with the morbid state of the anterior nerve-roots. The condition of the cell groupings here was as follows:—

The *postero-lateral group* of cells is most extensively degenerated.
The *central group* (Ross), together with the former, are the groups most generally affected in this region.
The *antero-lateral* and *medio-lateral* cells are also seriously involved, but less frequently so than the former two groups.
The *internal group*, although occasionally found degenerated, is far more frequently quite intact.

In the opposite hemisphere the conditions are very different.

The *antero-lateral*, *medio-lateral* and *postero-lateral* groups are almost invariably represented by numerous healthy well-branched cells.
The *central group* is frequently found to consist of shrunken and atrophied cells.
The *internal group* is occasionally found degenerated.

The more frequent condition in this region was a perfect development of all the lateral and inner cell-groupings, with perhaps slight disease of the central cells in one hemisphere as contrasted with an extensive implication of all the lateral groups, *especially the postero-lateral and often complete destruction of the central group* in the opposite hemisphere. Even in the healthier hemisphere, however, the marginal cells of the antero-lateral series are often swollen, globose and succumbing to a diseased process;

whilst in one preparation the postero-lateral group was represented by one or two stunted cells, and all the cells of the median group were absent. In *both hemispheres* the central group was frequently represented by mere shrivelled and formless relics of cells situated in the midst of deep stained sclerosed tissue, often with a large dilated and diseased vessel in this neighbourhood. In like manner the morbid process in the postero-lateral group was *always in advance* of the degenerative changes seen in the remaining cell-groups.

The features claiming chief attention, therefore, in the cervical region were as follows.

a. Atrophic shrinking of the cell-groups, chiefly of one hemisphere.
b. This atrophy most apparent in the central and lateral groups.
c. Morbid changes apparent in the anterior roots of this hemisphere.
d. Slight sclerosic shrinking of the corresponding anterior horn, causing diminution in bulk of the whole segment.
e. Limitation of such changes to the lower cervical regions, the cell-groups being more and more healthy higher up, until on a level with the third cervical nerve they are perfectly intact.

THE DORSAL REGION.

a. Posterior Columns.—As before mentioned, these columns were extensively diseased and consistence so fragile that good sections could not be made without extreme difficulty. Its structure was perfectly riddled throughout by numerous cavities—the perivascular tracts being converted into huge channels, here and there still further distended into oval or spherical dilatations arranged in moniliform series along their course. The membranes of the cord are especially thickened in this region, and the diseased condition of the vascular tunics suggests itself strongly as the origin of the further changes induced in the nervous tissues, since the lesion is decidedly more advanced along the *peripheral border* of the columns, and spreads inwards along the direction of the vessels. The columns of Burdach are much implicated on both sides, but least so in the right hemisphere; the posterior commissural zone is sclerosed to a minor extent, whilst the peripheral margin adjacent to the membranes (sub-meningeal zone) is converted into a zone of pure punctated tissue. In all cases alike it may be stated that the deepest and most advanced morbid change has taken place in this submeningeal zone; still further inwards, acute changes are seen in the presence of numerous swollen nerve-fibres, colloid bodies, and the extension of sclerosis along the vascular tracts; whilst in the post-commissural zone the vessels of this region have apparently set up a distinct focus of disease, the areas here being mapped out by numerous deep-stained patchy sclerosic islets and largely dilated nutrient branches.

The sclerosed tracts invariably followed closely the course of the diseased blood-vessels.—The columns of Goll near their meningeal extremity, have often large circular cavities excavating their structure, identical to those above described, and the nerve-tissue *immediately* surrounding such spaces was condensed, sclerosed, and deeply stained.

The posterior nerve-roots on either side are extensively implicated, being so greatly wasted that scarcely a solitary axis-cylinder is seen.

β. *Lateral Columns.*—The *direct cerebellar tract* is involved throughout nearly its whole extent, deeply stained punctated tissue occupying the interspace betwixt the diseased nerve-tubes spreading inwards from the meninges. Its distribution is symmetrical on both sides, but the sclerosic change has upon the left side invaded the *crossed pyramidal tract*, which here contains notably enlarged coarse vessels, similarly affected to those of the posterior columns. The lesion in the cerebellar tract is very distinct; to a certain depth pure fibrillated tissue is found, and deeper still, numerous islets of the same intervene betwixt wasted nerve-fibres and amongst dilated vessels. The implication of the cerebellar tract, however, is less extensive and intense than in the cervical cord. The crossed pyramidal tract has escaped implication on the right side. The changes in the left crossed pyramidal tract cannot be traced downwards in the lowest segment of the dorsal cord, nor yet are they seen at the commencement of this region.

γ. *Anterior Columns and Root Zone.*—The columns of Turck (direct pyramidal tract) were quite healthy. The anterior root-zones appeared in every respect free from disease, but the submeningeal zone was occasionally invaded from the membranes to a slight degree, and the tissue here was much degenerated and friable. The condition of most of the sections obtained was unsatisfactory to a determination of the state of the anterior nerve-roots, which must therefore be left undecided.

δ. *Anterior Cornua.*—A notable sclerosic change had affected the left horn of grey matter in this region of the cord. The grey matter had shrunk, so that its transverse diameter was much reduced, the left cornu being represented by a narrow tract of dense tissue one half the width of the opposite horn; at the same time, whilst undiminished in length, it was more pointed, having lost the more square form of the corresponding cornu of the other side (Fig. E). Its deeply stained condensed tissue contained the relics of diseased cells. Corresponding to this lateral shrinking of the cornu the whole hemisphere was flattened and narrowed laterally, in marked contrast to the rounded contour of the opposite segment, measuring in diameter 3·5 mm. against 4·5 mm. for the healthier hemisphere.

The various cell-groups, fused together as they naturally are in this region, and difficult to distinguish, are extensively atrophied and often entirely wanting; the remaining ones being either swollen, devoid of branches, and with nucleus indistinguishable, or pale, shrunken, and also without connections. The grey substance of the cornu was much diseased; often the site of enlarged perivascular channels, its texture friable and broken down. At the lower end of the dorsal cord the posterior commissure is completely severed from its right lateral connections by one of these large cyst-like cavities, whilst a similar lesion has divided the anterior commissure from the left segment of the cord (Fig. D). Such lesions appear to indicate extension of the disease to the anterior horns, chiefly along the course of the perivascular tracts.

THE LUMBAR REGION.

a. Posterior Columns.—The whole of this tract is the site of large dilated vessels, the lymphatic sheath implicated in like manner but to a minor degree. As in the dorsal region, the part most seriously involved is the "sub-meningeal zone," where the medullated tubuli have entirely disappeared, and to the depth of ·325 mm. a pure punctated fibrous tissue has replaced the nerve-fibre, and extends inwards along the course of the blood-vessels. To a still further depth of ·4 mm. the more acute stage of degeneration is evidenced in the large swollen contour of numerous nerve-fibres.

Patchy sclerosic areas are found, especially along either side of the posterior median raphe, as far as the posterior commissure. The medullated fibres which have especially escaped implication are those found at the *innermost extremities* of the columns of Burdach. The *internal radicular fibres* are observed in sheaves passing into the posterior cornu, apparently not involved to any very notable extent. The peripheral portion of the whole posterior columns is riddled by fissures and cavities, produced as in other regions of the cord.

β. Lateral Columns.—The invasion of the outer and more posterior tracts of these columns is clearly seen; the deep-stained sclerosed tissue extends forwards across the posterior nerve-roots, and implicates the *lateral margins* and the *crossed pyramidal tracts.*

Numerous large spider-like connective cells are spread over the field, and much punctated fibrous tissue intervenes between the medullated tubuli, which likewise exhibit the segmentation of their medulla as above described.

The medullated tract bordering the grey matter in these columns is invariably intact, but many of the nerve-fibres which pass from the lateral columns horizontally inwards towards the anterior cornua appear to be degenerated. In these latter fibres the *axis cylinder is often seen enlarged.*

γ. Anterior Columns and Root Zone.—The columns of Turck are healthy throughout their whole extent on both sides. A similar statement may be made respecting the anterior root-zone, with this limitation; that the more external bundles of *descending medullated túbuli*—in other words, the peripheral or "sub-meningeal zone," seems implicated! In this region a suspicious amount of punctated tissue is found pervading the nervous tracts, so as to appear like an exaggerated development of the peripheral cortical zone of neuroglia, deeply stained. It does in fact appear as though the greater part of the peripheral border of the lumbar cord has undergone more or less sclerosis in the *immediate neighbourhood of the investing membranes.*

The *anterior nerve-roots* are in all cases perfectly intact, and are given off in dense bundles, traced as clearly defined tubuli quite through the columns of the cord from the anterior horns to their point of exit.

δ. Anterior Cornua.—The various groups of cells were well represented and healthy throughout the lumbar enlargement, and in all respects the cornua appeared to have escaped invasion by the lesion seen in other tracts.

The Vesicular Columns of Clarke, &c.—Throughout the whole of the dorsal cord the region of Clarke's column was extensively degenerated. The tissue was deeply stained, friable, readily broken down in these sections, and invariably showed diseased cells. The latter were usually inflated and globose, often in the last stages of pigmentary degeneration, shrunken or wholly destroyed.

In the immediate neighbourhood of these columns the vessels were diseased and extremely coarse; whilst occasionally a huge perivascular cavity occupied the site of one of these columns, similar to those found in the posterior columns.

The central canal was, in all regions alike, completely choked up by proliferated endothelial elements.

EXAMINATION OF THE PONS AND MEDULLA OBLONGATA.

The degenerative change recognised in the posterior columns of the cord was traced upwards along the funiculi graciles and funiculi cuneati into their respective nuclei, the cells of which nuclei were diseased and shrunken. The inner series of arcuate fibres, passing from these nuclei forwards towards the olivary body, were apparently diseased.

The outer division of the restiform tract (restiform body proper) was perfectly free from disease, with exception of a marginal border representing the direct cerebellar tract, which latter column was notably diseased and distinctly mapped out at lower levels, as a deep-stained sclerosed bundle lying in front of the caput cornu, and at higher levels also where it covered the restiform column and the ascending root of the fifth nerve. The lesion in this cerebellar tract was similar in all respects to what was met with throughout the spinal cord, and both columns were affected to about an equal extent.

The ascending roots of the fifth, although close in the proximity of very advanced degenerative changes, *had wholly escaped implication*, and, traced upwards to the level of the emergent fibres of this nerve through the pons, were found to be perfectly normal.

Throughout the whole region of the medulla occupied by the inferior olivary body no other morbid change was detected than the following—

1. Degeneration of the nuclei of cuneate and slender funiculi.
2. Degeneration of the direct cerebellar fibres over the restiform tract.
3. Sclerosis and absence of cells from the *most external region* of the vagus nucleus on each side ("zellenarmer Theil des Vaguskernes" of Schwalbe).
4. Partial sclerosis of the "solitary funiculus" both in the regions of origin of the vagus and of the glosso-pharyngeal nerves.
5. Extreme varicosity of emergent fibres of glosso-pharyngeal around the solitary funiculus.
6. Extensive atrophy of inner arcuate series of fibres passing from the nuclei of the slender and cuneate columns forwards behind the olivary body.

With exception of the partial affection of the column of origin of the lateral mixed system of nerves (vagus, glossophar. and sp. access.) and implication of the outer region of the posterior vagal nuclei, no other cranial nerve, from the third to the twelfth inclusive, showed indications of disease either in their nuclei or emergent fasciculi.

Each individual nerve was closely examined and the surrounding areas of motor and tegmental tracts were alike normal. Both inferior olives were healthy in every respect.

EXAMINATION OF A PORTION OF SCIATIC NERVE.

In the fresh state the nerve was of far less firm consistence than in the normal condition, apparently containing much fatty tissue which gave it the aspect and feel of the degenerated sciatic nerve in certain advanced cases of general paralysis. Upon transverse section, the far greater area was occupied by a yellowish, spongy, adipose tissue, three nerve-bundles being seen separated widely by this intervening tissue. Through the centre of this adipose structure coursed two or three minute fasciculi, subsequently recognised as the remains of degenerated and greatly wasted bundles of nerve-fibres. The nerve hardened by chrome-salts had a greasy, unctuous feel, and a large amount of fatty matter had been set free from it in the chrome reagent. Sections taken from fresh and hardened specimens alike exhibited a great surplus of adipose tissue, the cells swollen with fat globules which likewise infiltrated the *epineurium* in all directions. The latter consisted of wavy fibrous tissue, with numerous fatty and degenerated connective cells; whilst larger and smaller oil-globules seen in osmic-acid preparations disposed along the course of these fibres, surrounding its blood-vessels also, and in some cases, penetrating the sheath of the fasciculi, lie disposed upon the outer aspect of the nerve tubuli betwixt them and the *perineurium*. Longitudinal sections showed the nerve-tubuli in most cases in a state of granular disintegration; the medullated sheath having a coagulated aspect, and containing within it coarse and small granules in great abundance. The axis-cylinders had undergone no change, and there was no strangulation of the inter-annular segments by the swollen nucleus.

The segmental nuclei were in most cases not visible, or very indistinct, and faintly stained by carmine. No varicosities of the sheath with proliferating and granular nuclei were observed, such as occurred all throughout the degenerated tracts of the cord.

At times the medullated investment appeared wholly gone, leaving the axis cylinders naked, but otherwise intact. The *endoneurium* in the less affected fasciculi presented no hyperplasia or notable change. In conclusion, the nerve throughout its course showed extensive granular disintegration of the medullated sheath of the nerve-fibres, with extreme fatty infiltration of the epineurium, and fatty engorgement and increase in the amount of the natural adipose tissue of the nerve.

W. B. L.

EXAMINATIONS OF THE OPTIC NERVE AND RETINA OF THE LEFT EYE.

BY EDWARD NETTLESHIP, F.R.C.S.

The diameter of the optic nerve itself (i.e. not including the outer sheath) is considerably less than that of a healthy nerve for at least half an inch behind the eye (the longitudinal sections do not extend further back). In a longitudinal (axial) section extending from the disc for half an inch backwards the following appearances are observed:—No nerve-fibres can be made out distinctly in the lamina cribosa, nor for a short distance beyond it, the lamina cribosa itself being much thickened by connective tissue, which has the same appearance as that of the normal lamina. The thickened lamina is convex towards the eye as it usually is in cases of swollen disc, but this change in the present specimen seems accounted for by the increase of tissue, without the separation of its fibres by œdema which is seen when the disc is swollen in neuritis. The nerve-fibre bundles begin to be apparent a little way behind the lamina, and become more and more so towards the brain-end of the specimen; but even at half an inch from the eye the nerve is almost as much shrunken, so that many nerve-bundles must probably be wanting.

In all the transverse sections examined, the fibrous trabeculæ between the nerve-bundles are decidedly thickened; this change, however, is far more marked near to the globe than in sections taken further back. There is a corresponding difference in the size and distinctness of the nerve-bundles, the nerve-tissue being scanty and ill-defined near the eye, whilst in the most proximal section these changes are almost confined to the peripheral bundles, and even then are present in a higher degree in some parts of the periphery than others, and a large number of the more central bundles show little if any change. The small blood-vessels in the thickened trabeculæ are full of blood, and their coats are sometimes thick. The intra-vaginal tissue of the nerve-sheath is considerably thickened in some, but not in all places.

The appearances described seem to point to atrophy having begun at the optic disc, or immediately behind it, and to its having been accompanied (or caused?) by a considerable increase of the connective tissue of the disc-floor itself. Whether the thickening of the trabeculæ further back is due to a primary hypertrophic change in the connective tissue, or is secondary to a previous atrophic process in the nerve-fibres, cannot be conclusively decided from this specimen, as the staining is very imperfect, and I do not know in what fluid the specimen was hardened. It may be pointed out that the evidence in this specimen of the changes having begun at the disc is in accordance with clinical observation.

In the retina, close to the disc, the nerve-fibre layer is much attenuated and composed of well-defined fine wavy fibres with oval nuclei, representing connective tissue or shrivelled nerve-fibres; no ganglion cells are visible, but the other layers, including the rods and cones, are much better preserved than is usual in post-mortem specimens, and look healthy.

I do not know the state of vision in this eye, but from the absence of visible nerve-fibres in the disc, it is difficult to suppose there could have been any sight.

In regard to the ascending changes, it would be interesting to know how long the eye had been blind, if it was blind.

E. N.

This patient presented the following characteristic symptoms of tabes dorsalis.

Ataxy.

Absence of knee-phenomenon, the vastus internus muscle responding to direct percussion.

Shooting-pains.

Giving way of the legs.

Difficulty of walking in the dark.

Numbness and anæsthesia.

Feeling of trunk constriction.

Diplopia.

Optic nerve atrophy.

Argyll Robertson pupils.

Delay in perception of painful impressions.

Bladder trouble.

Besides these, he had for some time complete loss of power in the iliaco-psoas group of muscles, and towards the close of life obstinate vomiting, with abdominal pain (gastric crises).

I have enumerated the symptoms to show that there can be no question regarding the nature of the case. The symptoms during life, confirmed by the appearances after death, point conclusively to tabes dorsalis. No doubt the case presented certain peculiarities. The short duration of the disease, which, if the patient's account is to be trusted, extended only (from the time of attack to death) over a period of twenty-seven months, is remarkable. The degree in which the blood-vessels of the cord are affected, as described by Dr. Lewis, is also unusual. The only case I remember to have seen comparable with it in this respect was that of a female, who was in the hospital a few years since under my care, with disseminated cerebro-spinal sclerosis of rapid progress. In her case there was granular disease of the kidneys. The blood-vessels of the cord were greatly thickened in their walls, the perivascular spaces very large, and there were extravasations of blood, in connection with the diseased vessels, so marked as to be visible by the naked eye on sectioning the cord.

As regards the appearances in the spinal cord presented under the microscope, I quite agree with the opinion expressed by Dr. Lewis. The lesions point, as he has described, to a peri-arteritis apparently originating in the soft membranes, and spreading inwards along the nutrient supply of the posterior columns. That the disease had not remained strictly confined to these columns but had spread, though in far less degree, to other parts of the cord, is what may often be seen in cases of tabes. The tendency to diffusion of this kind seems indeed to me to constitute a considerable difficulty in the way of unqualified acceptance of the hypothesis that the nervous elements are primarily affected. It offers far less objection, on the other hand, to the hypothesis of such a sequence of events as is already generally accepted in the case of disseminated sclerosis.

I do not propose to advance the opinion that the group of symptoms to which we give the name "tabes dorsalis" is always the result of arterial changes, spreading from the soft membranes to the posterior columns of the cord; but I am inclined to think that it is much more often so produced than is commonly supposed. A certain difficulty here presents itself. Can we allow the probability that a disease identified by the symptoms of which I have given a category as they were observed in the present case, is sometimes the result of changes in the connective tissue bringing about destruction of nerve tubes, and at other times originates in the nerve tubes and spreads to the connective tissue?

The idea entertained by authorities who are entitled to our greatest respect, Charcot and Vulpian, appears to be that the disease may be imitated, so to speak, in consequence of lesions originating in other conditions invading the anatomical district which is affected in tabes. Charcot, for instance, remarked in 1873, that certain lesions of the spinal cord primarily developed outside the posterior columns, might at a given moment invade these at a variable height, and produce, accidentally, as it were, some symptoms of locomotor ataxy. He went on to say that this was *not truly progressive locomotor ataxy.* Vulpian too[1] says, "This disease is primitive or secondary. In the latter case it is produced in the sequel of another affection or previous lesion of the cord. . . . But in these cases the symptomatic expression of ataxy is manifested but seldom with its complete characters, and in such a way that the diagnosis of this complication can be laid down with any certainty."

These cases of secondary origin are supposed therefore to differ materially from the primary disease. I cannot help thinking, however, as I have before suggested[2] that in enquiring into the history of many cases which, when they come before us, present all the characteristics of typical tabes dorsalis, we frequently hear a description of symptoms, often long past, which can only be referred to the occurrence of spinal meningitis, usually of a slight and strictly localised character. In the present instance, as will have been noticed, there was not a symptom wanting of the typical form of the disease, and the chief and perhaps sole peculiarity of the case was the rapidity of its progress to a fatal termination.

In connection with this point I would refer especially to the atrophy of the optic nerve. It is generally agreed that we know of no anatomical association which will explain this lesion as the result of a continuity of nerve elements. That it is not produced (as the sclerosis in the cervical region, for instance, is supposed to be produced) by extension of the irritative process along the nerve-fibrils is evident, both from the fact that it often precedes the spinal symptoms, and also because, as Mr. Nettleship has shown in the present instance, the disease affects the *periphery* of the nerve, and gradually ceases as the centre is approached. So that in the so-called secondary form this interesting peculiarity as regards the position of the degenerative changes in the optic nerve (their centripetal character) is exactly reproduced. Then again there is the transitory diplopia from which, as the history tells us, this man suffered. One cannot help feeling it extremely difficult to explain how it is that these two conditions, peculiar, I think, to this disease, can result from lesions so completely opposed in their origin as those which are thought to characterise primitive and secondary tabes—in the one case an irritative lesion of nerve-fibres leading to hyperplasia of neuroglia—in the other a disease of blood-vessels and neuroglia leading to lesion of nerve-fibres.

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[1] 'Maladies du Système Nerveux. Maladies de la Moëlle.' Paris: 1879.

[2] Loc. cit., p. 204.

Professor Adamkiewicz of Cracow, in a paper read before the International Medical Congress held in London,[1] expresses the opinion that the conception of tabes consisting in a primary degeneration of nerve-fibres in the posterior columns, has been founded on examinations of developed forms of tabes only. He had the opportunity of examining cases in an early phase, and then found the degeneration not diffuse, but involving the connective tissue corresponding to the course of blood-vessels. He made injections of the spinal cord, and ascertained that the signs of degeneration followed the course of the blood-vessels, and involved, 1. The posterior free margin of the posterior columns; 2. The posterior nerve-roots. 3. The septum of the posterior columns. 4. The border between the columns of Burdach and Goll: and lastly, 5. The tracts originating in the inner borders of the posterior horns and converging towards the posterior borders of the posterior columns. Upon the evidence that the beginning of tabetic degeneration coincides with the course of the blood-vessels, he founds the opinion that tabes is an interstitial degeneration of connective tissue. For it is the connective tissue, he remarks, not the nerves, which accompanies the blood-vessels. Tabes thus resemble cirrhosis of the liver, which is an interstitial degeneration proceeding from the connective tissue. This view of the interstitial nature of tabes is clinically of importance, as it explains, he says, the optic-nerve atrophy which frequently accompanies tabes. One has only to suppose that along with the blood-vessels in the cord, coincidently or frequently the blood-vessels of the optic nerves are diseased.

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[1] "Tabes ist eine interstitielle Degeneration des Bindegewebes in den Hintersträngen," 'Transactions of International Medical Congress.' London, 1881, p. 163.

This experience, although very interesting in connection with the present case, is of course by no means conclusive. Indeed it will need much evidence to shake the position which Vulpian defends by arguments of singular force and logical accuracy. But the question is evidently still *sub judice*.

It must be remembered that the vascular supply of the optic nerve passes from centre to periphery. Altered states of nutrition dependent upon disease of the vessels would probably therefore, I would submit, be experienced *first at the periphery of the nerve*, and gradually tend thence upwards. It may be conjectured that a similar explanation might possibly account for those peripheral changes in nerves described by Pitres and Vaillard in an important paper lately published.[1]

In the account of the autopsy it will be noted that a number of calcareous bodies was found in connection with the dural layer of the arachnoid. The presence of bodies of this kind has been before remarked in this disease. Vulpian more than once refers to it, and so also does Westphal, but without attempting, I think, to explain their nature.

It occurs to me that they may possibly lend evidence of some importance as to the relative *age* of the meningeal changes. The changes in the cord can hardly, I should think, have preceded these calcareous deposits in the membrane.

We acquire, too, some useful information from the clinical

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[1] 'Archives de Neurologie,' No. 17, 1883. (See 'Abstract' in the present Number of 'BRAIN.')

ON THE RELATION OF THE "AURA" GIDDINESS TO EPILEPTIC SEIZURES.

BY CHARLES E. BEEVOR, M.D. (LOND.), M.R.C.P.

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THE feeling of giddiness occurring as a "warning" or aura before epileptic seizures has been long known, but as far as I have been able to ascertain I have failed to find, in the writings on the subject, an account of any association or relation between the character of the giddiness and the commencement of the ensuing seizure. I shall endeavour in this paper to show that there is some relation between the direction of the apparent movement of the patient and objects surrounding him and the initial rotation of his head at the commencement of the fit.

I think it probable that this apparent movement and the initial rotation are usually in the same direction, or, in other words, that objects appear to move, and the patient feels himself to turn in the *same* direction as his eyes and head are actually drawn in the commencing tonic spasm.

In Vol. III. of 'BRAIN,' p. 192, Dr. Hughlings-Jackson has contributed a valuable paper "On Right or Left-sided Spasm at the Onset of Epileptic Paroxysms," and in speaking of vertigo *in the sense of there being apparent displacement of objects to one side* as occurring in those paroxysms in the first part of which the convulsion is right-sided, Dr. Hughlings-Jackson does not commit himself to any fixed conclusion on the matter. He writes: "I dare not state it as a fact, but my impression is," &c. He dwells on the difficulty of getting trustworthy information from the patient or his friends as to the side on which the initial spasm began or preponderated at the onset of epileptic paroxysms. He mentions various ways in which the terms subjective and objective are used by medical men, and states how he himself uses them. Admitting and illustrating that the division is arbitrary, he groups vertigo, colours before the eyes, and noises in the ear as crude developments of objective sensations; he groups smell, taste, and the sensation referred to the epigastrium, as crude developments of subjective sensations. Speaking of the so-called "intellectual aura," or, as he prefers to call it, "dreamy state," he says that, if there be also a crude sensation, it is nearly always one of smell, taste, or it is the epigastric sensation. A crude sensation of taste, is very rare, but movements of chewing or tasting are not uncommon with the "dreamy state," and are, he thinks, indirect consequences of discharge of central gustatory elements. With the "dreamy state" there may be vertigo in the sense of the patient himself seeming to turn to one side, or there may be "alteration" in the distance of objects in front of him; there is rarely, he thinks, along with the 'dreamy state" vertigo in the sense of apparent displacement of external objects to one side.

Dr. Mercier has described a case of epilepsy in the last (October) number of 'BRAIN' in which the patient, who had attacks beginning in the left side, experienced the sensation of vertigo in the form of feeling herself turning from left to right, accompanied by noises in the ears. In this case he finds that the vertigo and other sensations felt by the patient were of the objective order, and yet were associated with spasm commencing in the left side, which, as he states, happens to be the wrong side according to the hypothesis that objective sensations are usually associated with attacks beginning on the right side.

I have lately, in the out-patient department of the National Hospital for Paralysed and Epileptic, been investigating the cases of epileptic patients complaining of giddiness before a fit. The word "giddiness" may be taken to mean the apparent movements of objects round a person, or the feeling of the person himself moving round, or these two combined.

The cases, which I found presented the "aura" of giddiness, are seventeen in number. In none of them does the patient state that he feels as though he went the opposite way to which the objects seem to him to move. In eight cases they complain of feeling themselves and also external objects rotating round from right to left, and nine feel themselves and objects moving in a similar way from left to right.

I have endeavoured to ascertain whether the direction of the apparent rotation of objects and of the patient himself could be found to have any relation to the side on which the initial spasm of the epileptic seizure took place, or, in other words, to find out, in those cases, where the fit begins by giddiness and the feeling of revolution of the patient and objects in one direction—say to the right,—whether the attack begins with actual rotation of the patient's head to the right or to the left side, and whether the spasm begins in the right or left hand.

I think that great stress is to be put on this point (the initial rotation of the head) as a means of determining the starting-point of the fit, for in ordinary bi-lateral epileptic fits it is often quite impossible to find out from the friends, which side of the body is the more convulsed during the clonic stage, whereas the rotation of the head, which is usually present, can be more easily recognised by the friends, especially if they are requested to observe it.

In the above cases in which the vertigo occurred from left to right, in five the fits have commenced on the right side. Of these one patient, who complains of giddiness with apparent rotation of herself and objects towards the right, was an in-patient in the hospital, so that the nature of the attacks was carefully noted, and it was seen that the fit began with turning of the head and eyes towards the right; she was then convulsed on both sides of the body, and I noted immediately after the fit that the right knee-jerk (patellar tendon reflex) was more marked than the left, and that ankle-clonus was obtained better on the right than on the left (see "Knee-jerk after Epilepsy," 'BRAIN,' April 1882), thus showing that the right side of the body was more affected than the left. In another case with a similar warning of giddiness (this patient after another fit has said that she alone seemed to move to the right, and not objects), the friends state that the head turns to the right, the right arm is drawn up, and the patient is convulsed on the right side more than on the left. Another patient states that she feels that her mouth is drawn to the right, and that she experiences apparent rotation of herself and objects towards the right; she is also convulsed more on the right side.

I may here say that I have not any record of, what I may call, right-sided vertigo, accompanied by "subjective" sensations, but this is a point on which I cannot speak with any certainty without further data.

In all the cases where giddiness occurred with apparent movement of the patient and his surroundings from right to left, the fits have begun on the left side of the body, and in most cases the head has turned to the left in the initial tonic spasm. One patient volunteers the statement that he feels the left corner of his mouth to twitch, and, according to his friends, at the beginning of the fit his head turns to the left and his left arm is drawn up. Another patient has repeatedly told me that she and the room seem to go round to the left; this is preceded by a sensation of sickness, shooting from the epigastrium to the head. Sometimes, however, she alone feels herself to go round, and not the objects around her, and describes herself to be "so lost" as not to be able to see any objects moving. Her fits begin with rotation of the head to the left. In addition to the left-sided vertigo, another patient feels "funny" and giddy, and as she turns to the left she has the sensation of seeing balls of red, yellow and green in the order mentioned. Her fits commence by the head turning to the left and drawing up of the left arm. This patient, moreover, is very spiteful *before* the fits. Again, a patient had the first fit after a blow on the right occiput; he has now tenderness to percussion on that spot, and sometimes experiences pain there for some days before a fit. He has giddiness with apparent rotation of himself and objects to the left, and his fits begin on the left side. Another patient feels "lost" and faint with the double feeling of left-sided vertigo with actual rotation of head to the left.

Altogether there are twelve patients in which the apparent rotation of themselves and objects is in the same direction as the initial clonic spasm, seven of these being to the left and five to the right. Besides these there are four patients, who differ from the foregoing in the relation of the direction of the apparent rotation to the initial spasm. These four feel the rotation of themselves and objects to the right; one is described by his friends as being convulsed "all to the left;" another has his head drawn to the left and "struggles round to the left side;" a third case has the sensation of objects and herself turning to the right, and her friends state that the head turns to the left, that the left arm is drawn up more than the right, and the left side is convulsed more than the right. A last case, who is sure that objects and herself seem to go to the right, has left-sided fits, beginning with tingling and drawing up of the left foot, her head turns to the left, and in the fits she is convulsed left more than right. These last two patients are the only ones which entirely oppose the majority of the cases, in that right-sided vertigo is associated with a distinct left-sided spasm, the other three opposing cases not being in any way so certain. I am unable at present to give any explanation why these patients differ from the majority of the cases. One other patient has the sensation of apparent movement of himself and objects from right to left, but I have not yet obtained a definite account of the character of his fits.

In the above cases it is very difficult to be absolutely sure of the facts, when one has to depend so much on the statements of patients and their friends; and as spontaneous statements are worth very much more than answers to questions which are more or less leading, I have avoided as much as possible asking questions which would prompt the patient to give answers in harmony with one's preconceived notions. It is sometimes, however, almost impossible to avoid assisting the patients in their answers; as, for instance, a patient volunteers the information that he feels giddy before a fit, but if he is asked what he means by giddiness, he answers, as though the word giddiness expressed all that could be required or be possible to know, and he is quite unable to explain himself further; on this I usually ask whether he means by giddiness the feeling of faintness or whether he experiences any movement, when perhaps he will jump at the question and say immediately that he swims round or the room seems to go round; I then point to the window and ask which way it would go in his giddiness, and also make him put himself in the direction in which he would feel himself to be moving.

Sometimes, however, patients, in answer to the question, how they know that a fit is coming on, will say at once, "The room goes round," or "I go round and round," and then the direction can be ascertained without any fear of putting prompting or leading questions.

Notwithstanding the difficulty of proving the correctness of the patient's statements, I think that the large number of seventeen cases, all of which agree in stating, that they and objects appear to move in the same direction—to the right or to the left—must make it highly probable that their statements are true, and that, when patients experience giddiness and the movement of themselves or external objects at the onset of an epileptic fit, they, as a rule, feel to go in a definite direction, and probably objects seem to them to go the same way as they themselves; and further, that in most cases the primary tonic spasm does begin in the corner of the mouth or in the hand and arm on the *same* side towards which the apparent movement of the patient and his surrounding objects takes place. Some of the patients' friends have answered so decidedly my questions, as to which side or part is first affected in the fit, that I cannot but believe that their answers are correct.

I think I ought to mention, that in these cases I have referred to the feeling of giddiness associated with that of movement, either of the patients themselves or of external objects, in a horizontal direction to the right or to the left. I am therefore not referring to those cases, where the patient feels faint or sick, and calls that giddiness, or, on the other hand, to those cases where the patient describes that, as the fit begins, he merely feels his head being drawn round, to one side or the other, but without any giddiness. This feeling is similar to the sensation of the arm and hand being drawn up at the commencement of a fit, and may be unattended with any feeling of giddiness, though of course the two conditions may and do exist together.

In some of my cases the patients express different opinions as to whether the whole of their bodies or only a part seems to move. In some cases of giddiness the head alone feels to turn to one side while the rest of their body does not seem to them to move. I have not as yet been able to get any definite data in these cases, or to find anything to account for the whole body, or the head only, being affected with the apparent movement.

I should state that none of the patients mentioned above have complained of deafness or noises in the ears accompanying the giddiness, and therefore do not come under the class of cases mentioned by Dr. Ormerod in his paper "On Epilepsy in its Relation to Ear Disease," published in the number of this Journal for April 1883.

In connection with the subject of giddiness and movement of the eyes, I would refer to two cases of auditory vertigo, recorded by Dr. Hughlings-Jackson, one of which was published in the number of this Journal for April 1879. In this case the patient's eyes moved during a paroxysm in jerks towards the right side, and he described external objects as seeming to him to move from left to right—in fact, in the same direction as his eyes jerked. A communication from Prof. Donders was read at a meeting of the Ophthalmological Society in 1881 commenting on this case, but as space will not allow me to enumerate his various arguments, I would refer those who are interested in the subject, to vol. ii. p. 213 of the Ophthalm. Soc. Transact.

The other case, which was read by Dr. Hughlings-Jackson at the same Society, in January 1883 (Ophthalm. Soc. Transact. vol. iii. p. 261), was that of a woman who suffered from disease of the right ear, probably set up by infantile otorrhœa, and in whom pressure on the right tragus caused giddiness and a tendency to fall to the left side, and at the same time the eyes moved slowly to the left and came back with a jerk to the right. It was particularly noted that the patient described the apparent movement of external objects from right to left to occur at the moment that the eyes were seen to move slowly to the left, and not when they jerked quickly back again to the right.

Whatever may be the explanation of these two cases, and what bearing they may have on the subject of this paper, I have mentioned them for their great interest, and with the hope that they might support the cases mentioned in this paper. The difficulty is increased by the fact, that in the man the apparent movements of external objects occurred with the rapid jerks of the eyes, and in the woman they occurred with the slower movements of her eyes and not with the rapid jerks. Now in epilepsy one of the most marked motor symptoms at the beginning of a fit is the conjugate rotation of the eyes and head towards one side, and this side—right or left—is the one on which the patient is subsequently in the clonic stage the more convulsed. It is, therefore, rather remarkable, I think, that so many of the cases in this paper—twelve out of seventeen—have felt themselves and the room to go round in the *same* direction as that in which their eyes and head have been seen to turn as the fit began. In fact, these cases seem to agree with Dr. Hughlings-Jackson's first case, concerning which, to use Dr. Jackson's own words, "some of his friends told him that the eyes 'could not' have been moving in the same direction towards which the objects were displaced, and that he must have observed wrongly." I had hoped that the cases I have seen might help to confirm what Dr. Jackson described in his two patients, or perhaps only in the case of the man.

If the statements made by these patients and their friends are true, that in the majority of the cases the apparent movement of the patient and his surroundings is in the same direction as the initial spasm, it is very difficult to explain why this should be so, and I cannot pretend to be able to give an explanation.

I cannot see why there should be any reason to presuppose that objects *ought* to move in the contrary direction to the patient's head and eyes, or why he ought to feel himself going in the opposite direction in which the room seems to go. Our common ideas of giddiness can be produced by prolonged movements of ourselves or of objects before our eyes, and it seems to me that they are different to the feelings of giddiness before epilepsy which are of central origin.

To take two examples of induced giddiness: If one turn round on one's heel several times from left to right until giddiness is produced, it is said that the room goes in the opposite way, but in reality, as one turns from left to right, the room seems to rush past one from right to left, and when one stops turning, the room still seems to move in the *same* direction, viz. from right to left, while the experimenter feels himself going in the same direction in which he originally turned himself. The feeling of nausea, I find, is much increased if the eyes be kept looking on the ground, the head being bent down, so that the chin touches the sternum and the vertex looks forwards; and if the person then turn himself from left to right, and then on stopping raise his head to the erect position and look at the opposite wall, the phenomenon quoted by Dr. Bradbury ("On Vertigo or Dizziness") from Purkinje is seen, and the movements of the floor are transferred to the wall, which appears to rotate in a direction opposite to those of the hands of a watch; thus seeming to show that the impression of the movement of the floor is reproduced in the same direction on the wall.

On the other hand there is the phenomenon first noticed by Brewster and Addams, referred to in 'BRAIN,' October 1880 ("Optical Illusions of Motion," by Dr. Silvanus Thompson), that when one looked out of a railway-carriage in motion at the ground for some time, and then closed the eyes, undistinguishable forms and patches of light were seen to go before the eyes in the opposite way to which the ground had appeared to be rushing past the eyes.

Addams noticed that when a waterfall was looked at for some time and the eyes were suddenly turned to rocks close by, these appeared to move upwards in a direction opposed to the waterfall. These two latter experiments have been explained by an "unconscious slipping of the inferior and superior recti muscles," but they are more probably due, as Dr. Silvanus Thompson thinks, to a complementary reaction of movement to the eyes, which are tired by a continual movement in one direction.

In the case of turning oneself round on one's heel, the auditory apparatus cannot be excluded from having a share in the giddiness produced, but whether they could counteract and modify the ocular vertigo, I am unable to say. Unless we can explain these two different results I do not see how we are to presuppose any definite direction in the apparent movement of the patient or his surrounding objects when he feels giddy before an epileptic seizure.

I shall endeavour to pursue the subject further, as I am sure there is a great deal more to be found out in these complicated phenomena.

NOTE ON A CASE OF ANÆSTHESIA.

BY H. B. DONKIN, M.B. (OXON.), F.R.C.P.,

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THE greater difficulty of establishing the existence and assessing the value of modifications of sensation than of motion as symptoms of nerve-disturbance is of course obvious. Not unfrequently, however, this point is overlooked in reporting cases, and even in drawing important inferences from alleged accounts of sensory abnormalities. At first sight it seems clear that heightened or lowered sense-functions in human beings, depend very largely for the evidence of their existence on the statement of the patient: or in other words are to a great extent "subjective:" while motor disturbance, whether it be in the direction of spasm or paralysis, is much more open to observation or demonstration, and may generally be regarded, with certain limits and precautions, as an "objective" symptom. We all know that a great power of control over the expression of pain or pleasure is possessed by many persons: and that, within the bounds of obvious nervous health, there is often shown a capacity of bearing with apparent indifference the infliction of what most would undoubtedly regard as pain, not to mention the extreme sensibility of others to stimuli which most would ignore as indifferent. Instances of this are too numerous to necessitate quotation. Notwithstanding these facts, it is not uncommon to find the patient's mere statement of heightened or lost sensation, in certain conditions, or after certain applications or remedies, received even by skilled investigators as of equal symptomatic value with demonstrable alterations of motility. Some of the French writings on the neurology of sensation, especially those which deal with the phenomena of anæsthesia as related to "metallo-" and "magneto-therapy," are notable instances of apparent neglect of the possible vicious intervention of this fallacy in the experiments which they detail and regard as of so great importance.

At the bottom of this constantly occurring confusion in medical and psychological writings, as, indeed, at the bottom of a vast majority of controversies in general, lies a want of clearness of conception of the thing to be expressed, and still more a want of precision in the terms used to express it. In psychology there is an ever-recurrent danger of using physical and psychical terms interchangeably, and in a slip-shod, and sometimes almost disingenuous manner. It would indeed be well in the interests of true physiological psychology if the term consciousness could be altogether abolished, for thus a large amount of metaphysical haze would be dispelled. But perhaps this desirable expurgation of our terminology cannot be made. The term *sensation* though in its physiological application to the sensory nerves, for instance, it stands, or should stand, for the conductivity of impressions from the periphery to an ascending séries of centres, is frequently confused with the results of this nervous action as expressed in the feeling (or the consciousness) of the individual. Centripetal nerve action should be kept in our minds quite apart from " conscious " sensation which is the result of some metabolic activity in the higher centres; and if we must use any derivatives of the familiar words " sense " and " conscious " in this context, let us never lose hold of the fact that unconscious sensibility, or unfelt centripetal nerve action, is one thing, and conscious sensibility, or true " feeling," is another. The occurrence, then, of apparent anæsthesia may be regarded as possibly due to an interference with any part of the centripetal nervous apparatus, from the periphery up to the highest sensorial centres.

It should be the primary object in endeavouring to establish or fix the import of any alleged case of anæsthesia, for instance, to eliminate as much as possible the volitional element. Reported anæsthesia may be due, first, to shamming; secondly, to a preconceived mental impression or expectant attention; thirdly, to a mixture of these two elements; and fourthly, may be quite independent of these agencies, and strictly called physical. How the tendency towards shamming pure and simple is often allied with Hysteria, and how difficult it is sometimes in a given case to disentangle the passive from the volitional factor in the symptom we are studying, is familiar to all who are conversant with nerve-disorders. It is certainly most incorrect and culpable to put down the bulk of hysterico-nervous symptoms to shamming: many of the most distressing expressions of hysterical disease being as completely beyond the control of the will as the pain felt by most ordinary persons on the extraction of a tooth. But it is a wide jump from this position to the belief that mental influence on the body plays but an insignificant part in the production of abnormal sense-impressions. Many observers have now placed it beyond doubt that, in cases where mere shamming could be with the greatest probability excluded, expectant attention can be substituted to a very great extent for the older and vague belief, of late galvanized into a sort of pseudo-vitality in France, in some special influence of metals and magnets on the sense-functions. The experimental methods of "difference" and "concomitant variations," by the use of widely varying applications, in competent hands, have demonstrated the mental factor in many of these cases, thus adding only fresh instances to the numbers already accumulated in such writings as those of Braid, Carpenter, Hack Tuke and others on the influence of the mind upon the body. Yet, erroneous as it may be to refer such cases as many of those reported by M. Charcot and his school to shamming rather than to expectant attention, it is not uncommon to meet with examples of the great difficulty there is in separating these two elements, or, indeed, in establishing the fact of genuine anæsthesia at all; and, when we think we have established it, of coming to a probable conclusion as to the presence or the proportion of controllable elements in its production. I give the following condensed account of a somewhat remarkable and interesting case in illustration of this difficulty.

Robert Warne, aged nearly 13, was admitted under my care at the East London Children's Hospital on October 18, 1882.

The account of his previous condition, as obtained from the mother, was that he had had measles, scarlet fever, and whooping-cough, and had suffered much from worms; no diphtheria, or other complaint. Three years before admission he fell down stairs and hurt his chin, since when, his mother says, he has from time to time complained of his head. A fortnight before admission he said he had a pain in his head, which spread down his back and right side, and he lost power in his right arm for quite five minutes. Five days before admission he is said to have had a fit in the night: his "limbs shook and his skin was discoloured." His right thigh also was said to be swollen, and he did not get up next day.

A few days before admission he suddenly fell down, as he was carrying a pail of water. He has never lost his senses, it appears; though he falls down sometimes and feels very faint.

The family history shows that his father has occasionally "fits," which are apparently epileptic.

On admission, the boy appeared to be quite well. There was no complaint or evidence of any motor disturbance; nor did a careful investigation of his organs and functions show anything abnormal beyond what follows.

The only complaint he makes is that of some pain and tenderness on the outer surface of the right thigh, and complete loss of feeling in his thumb. He says he sometimes sees everything red.

Examination showed apparently complete anæsthesia of the skin of the thumb on both aspects: the area involved being exactly limited by a line passing over the carpo-metacarpal joint. The pain in the thigh was not accounted for by examination, and soon passed off entirely. Over the rest of the body the sensation appeared to be good. The tests repeatedly used to the insensitive thumb, and often when the boy was off his guard, were pressure, pricking, pinching, the approximation of a lighted match to the skin, thrusting a needle under the nail and the use of the faradic current.

Subsequent and frequent examinations up to the end of November showed nothing fresh, except that it was thought that he was somewhat indifferent to ordinarily painful sensations over the body generally; though he felt the slightest touch, he said that pinching and pricking did not hurt much. There was possibly some little sensibility to the faradic current in the thumb—especially, as it seems, at the tip, but this phenomenon was not easy to establish. This possibility rested only on a slight twitch of the thumb, occasionally, when the current was applied. The boy denied feeling it. He had a slight discharge from his right ear, with some pain which soon disappeared. He was out of bed every day after admission, making no complaint.

On December 5th he complained of headache, and did not get up. The temperature remained normal. On the 6th he said he had pains in the lower part of his legs. On the 7th there appeared, on examination, to be perfect anæsthesia of both legs, from the soles of the feet up to a line one and a-half inches above the upper margin of the patella. This limit of anæsthesia was repeatedly verified afterwards, the boy being carefully blindfolded, and was tested by the application of all the methods above mentioned of exciting sensation or of inflicting pain.

The boy could walk well, and could stand in any position with his eyes shut without staggering. He said he could not feel the ground at all when walking or stamping; but when the hand was placed under his foot as he stamped, he said he could feel his thigh going up. His knee-jerk was somewhat excessive; no ankle-clonus. The hearing through the skull was good on both sides. On the right side the hearing by the meatus was impaired. He had been subject to a discharge from this ear, off and on, and the tympanic membrane was ruptured. Sight quite good. Colours and objects were accurately distinguished: and the optic disks were healthy in appearance. As to *smell*, he said, being tested, that "Eau de Cologne" and "Jockey Club" smelt nice in the left nostril, nasty in the right; and he stated that he could *taste* salt, &c., only on the left side of the tongue anteriorly, not at all on the right. Sensation to pricking was good all over the tongue.

This case was informally shown for me, by the kindness of a colleague, at a meeting of the Medical Society on Dec. 18, 1882, in order that others might verify the symptoms, which it was thought might disappear as suddenly as they had appeared. The conditions reported above were then ascertained to be present.

On Jan. 3rd, 1883, the boy, still in hospital, remained the same in every respect: a blister was applied to his right thumb, with no effect on the anæsthesia. He was then sent home.

On a visit to the Hospital on Jan. 28, it was found, by the same tests as before, that he had now loss of sensation on the ulnar side of right hand and little finger. This area of anæsthesia extended half-an-inch on to the back and palmar aspect of the hand.

On Feb. 8 I saw him again. He said that since the 5th he had had giddiness and headache, and felt as if everything were turning round him. On the 2nd, when lifting his arm to comb his hair, he had pain in left shoulder preventing him from doing so. On the 3rd, he says he could not raise his arm. The pain is now less. Anæsthesia the same as before.

In the month of April, 1883, I gained admission for this boy into the London Hospital, in order that his condition might be observed by Dr. Hughlings-Jackson. On the day of admission, as appears from the notes kindly sent us by the clinical clerk in charge of the case, the condition was the same as has been already reported. On the 14th, "the patient stated that the anæsthesia of the little finger had disappeared."

"On the 16th a strong faradic current was tried on legs, one pole being placed on his spine and the other on a portion of the anæsthetic area of one leg. Though he said at first that he did not feel it, it was evident by his expression that he did, and that he was trying to bear it. On slightly increasing the current his fortitude gave way, and he shouted out with the pain; which, however, on being questioned, he referred to the back and hip of the same side."

"On the 17th, he stated, the first thing in the morning, that sensation had returned in his legs; and, being tested with the æsthesiometer, sensation was found to be perfectly normal in the previously anæsthetic areas. The faradic current was then tried on the thumb. At the first application he declared that sensation had suddenly returned to that region. One minute previously, according to his statement when the æsthesiometer was used, the anæsthesia was as complete as on admission. (On testing his sense of smell and taste, soon after admission, they were found to be quite normal.) In speaking of the recovery of sensation in his legs, he said, the next morning, that he was suddenly awakened in the night with shooting-pains in his legs, and these soon left; a feeling of soreness remaining, and sensation being restored."

The case was said to be regarded from the first by Dr. Jackson as one due to "constructiveness."

The result of this case in perfect recovery, in circumstances such as have been detailed, certainly points with some plausibility to shamming as its cause; or, at least, as an element in its causation; and it might be supposed that the boy preferred residence in the Children's Hospital, where he had boys of his own age to play with, and, making himself useful, was a favourite in the ward, to going to school or being in the London Hospital. Yet, it did not appear in any way from his history that he disliked school or home; he did not seem anxious to stay in hospital, or to be considered as an invalid; and there was no evidence of any moral depravity or of hysterical tendency. The boy appeared in every way well and normal, with the exception of his apparent anæsthesia. And, it must be remembered too, that, with the hypothesis of shamming, we have to take into consideration the undoubted and often established fact of there having been frequently no signs or expression of sensation, on the application of what to most of the physically bravest men would be simply torture, and the long remaining exact limitation of the anæsthetic areas, as established by careful and repeated testing, when the patient was blindfolded, and with many other precautions. The anæsthesia of the legs was especially unvarying the whole time he was under observation at the Children's Hospital. On admission to the London Hospital, observations made to map out the area of anæsthesia of the thumb were said to be incongruous and contradictory, and "certainly to point to constructiveness."

If the case may be regarded as one of malingering, the absence of obvious motive is not so interesting as the complete control shown over the expression of suffering; a fact, if it be a fact, of enormous importance in studying the symptomatology of nervous disease, and in connection with all alleged cases of somnambulism, hypnotism, metallo-therapy, &c.

The theory of *expectant attention* will not satisfactorily cover the case, as all the symptoms were spontaneously complained of by the patient himself.

The elimination of the volitional element altogether leaves the case as an obscure one of organic or more probably, seeing that it speedily recovered, of functional anæsthesia. The distribution of the anæsthesia is, however, not after the pattern of recognised cases. It may occur to some that the application of the faradic current had some specific agency in restoring the sensation, especially as there was some little doubt as to whether, from the first, he could not feel the current when applied to his thumb.

Much of the interest of the case lies in the incongruity of the apparent physical phenomena with the theory of the moral obliquity of the patient. If this boy was shamming anæsthesia when he preserved a perfectly passive countenance as a needle was thrust under his nail, what test shall we apply for the numerous cases of alleged anæsthesia which we see and hear of in neurological cliniques, especially those of Paris? And, on the other hand, we may almost raise the question, what was the sensory condition of the Christian martyrs at the stake, or of the Spartan boy with the fox concealed in his bosom?

It seems on the whole more reasonable to conclude, in the present state of our knowledge, and keeping in view the salient facts of the case, that there was really a want of *conscious sensibility* in this boy, due either to a torpid condition of the sensitive nervous plexuses in those regions of the periphery where stimulation failed to produce its usual effects, and therefore to suspended centripetal conductivity, or to a disordered condition of the sensorium. The former hypotheses appear most probable, when we consider the distribution of the anæsthesia over both legs, the absence of any further abnormal symptoms referable to the cerebrum and the rapid recovery on the application of a strong faradic current. And though other so-called hysterical phenomena were not apparent, the case may perhaps be referred to the same category as some of the sensory disturbances so often seen in confessedly hysterical subjects.

It may be remarked, before leaving this case, that the boy complained of feeling pain in his legs both at the onset and before the disappearance of the alleged anæsthesia, and moreover that he appears to have been the probable subject of some form of epilepsy, especially when his father's attacks of "fits" are borne in mind. I cannot attempt a further explanation, but I find it hard to believe that the theory of shamming can explain the case away.

In connection with the subject of modified sensations in relation with mental states, the following case, quoted in Dr. Carpenter's work on Mental Physiology, from a Paper by the late Professor Hughes Bennett on the "Mesmeric Mania of 1881," is of some interest. A butcher was brought into the shop of a druggist, labouring under a terrible accident. The man, in trying to hook up a heavy piece of meat above his head slipped, and the sharp hook penetrated his arm, so that he himself was suspended. On being examined he was pale, almost pulseless, and expressed himself as suffering acute agony. The arm could not be moved without causing excessive pain, and in cutting off the sleeve he frequently cried out; but when the arm was exposed it was found to be quite uninjured, the hook having only touched the sleeve of his coat. "No evidence," says Dr. Carpenter, "could be stronger than that afforded by the almost pulseless condition of the patient, as to the reality of the severe pain which he experienced: and yet the pain entirely arose from his mental conviction that the hook had penetrated the flesh of his arm." This case may be compared with the alleged "sensitiveness," to what was called "odylic force," reported long ago by Reichenbach in connection with his experiments with magnets on human beings.

Clinical Case.

CASE ILLUSTRATING CEREBRAL LOCALISATION.

BY JOSEPH WIGLESWORTH, M.D. (LOND.)

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THE following case, in which permanent partial left hemiplegia, and amnesic aphasia were associated with lesions of the right paracentral lobule and left temporo-sphenoidal lobe respectively, appears to be of sufficient interest to justify its publication; and I am indebted to Dr. Rogers, under whose care the patient was, for permission to publish an account of it.

The patient was a female, aged 71, who died in Rainhill Asylum on the 31st of August last, having been an inmate of that Institution for four and a half years.

The only history obtainable was to the effect that she had had two hemiplegic attacks previous to admission. Her condition was pretty uniform during the whole time she was under observation; but it must be mentioned that the hemiplegia—to be shortly described—though never absolute, was most pronounced on admission, and that subsequently a certain small amount of improvement took place. Her condition as manifested at the commencement of the present year may be summed up as follows:—She was a heavy, big-boned woman, with hypertrophied heart, degenerated arteries, and signs of chronic Bright's disease. She had partial left hemiplegia; the arm was practically useless, though she had some slight amount of power in it; the fingers were bent into the palm, and the arm was kept permanently flexed; it could, however, be straightened: the leg was less paralysed than the arm, and patient could walk without assistance, though she did so with a decided limp, and her gait was manifestly insecure; the left leg was distinctly wasted as compared with the right, measuring round the most prominent part of the calf one and a half inches less than on the right side; knee-jerk distinctly exaggerated on the left side; no ankle clonus; no distinct facial paralysis. In addition to this hemiplegia, the patient had great disorder of speech. Her power of articulation appeared unimpaired, and she could articulate some words correctly; but when she attempted to express her ideas, her speech became an unintelligible jargon, in which, though individual words could be deciphered here and there, the incoherent jumbling together of single syllables seemed to form the most prominent feature; when talking rapidly her speech was absolutely undecipherable, not one word she said could be made out; she not unfrequently uttered this gibberish with great volubility. Her mental faculties appeared to be gravely impaired, though it is possible that, owing to the disorder of speech, she got credit for less intelligence than she actually possessed. As a rule, she sat very quiet, seldom attempting to speak unless addressed, and was in general tractable enough; if, however, her usual habits were in any respect interfered with—if, for instance, she was put to sleep in a strange room or bed—she got very restless and excited, and chattered away very volubly, though, as above stated, her speech was quite unintelligible. She clearly understood some things that were said to her; but the impression conveyed was, that her mental faculties were much impaired, that she was, in fact, considerably demented.

On the 25th of August last she was observed to be not looking well, and on examination of chest well-marked pericarditis was found to be present, and she had one-third albumen in her urine; her breathing became embarrassed, and she appeared to be suffering, but made no attempt to complain of anything; she gradually sank and died, as before mentioned, on the 31st of August.

The autopsy was performed on the same day, four hours after death, and the following is an abbreviated account of it.

Cranium.—Skull-cap a little thick; dura mater very adherent to it; three or four ounces of fluid in subdural space. Arachnoid and pia mater a little thickened, everywhere stripped very readily; considerable excess of subarachnoid fluid. Gyri generally somewhat wasted.

Right Hemisphere.—The gyrus fornicatus for the posterior two-thirds of its length had completely disappeared, as also the lower half of the paracentral lobule; these parts had evidently softened down and been absorbed, their position being occupied by a thin membranous bag containing fluid—they had, in fact, been converted into a cyst; the surrounding brain-substance was ochre-stained; the upper half of the paracentral lobule was also superficially softened.

The supra-marginal gyrus was also much atrophied and ochre-stained.

Left Hemisphere.—The middle of the temporo-sphenoidal lobe was occupied by a cyst with ochre-stained margins similar to the one above described; this involved the upper part of the middle temporo-sphenoidal gyrus, and the middle third of the superior temporo-sphenoidal gyrus; these parts were completely destroyed, and the white matter around, for some distance, was softened.

Cortex of average depth; striæ fairly distinct. Consistence of brain good. *Basal ganglia perfectly normal, as also were the crura cerebri, pons and medulla.*

Brain (immediately after removal) weighed 1064 grammes.

Right Hemisphere	896	grammes	stripped of membranes.
Left	417	,,	
Cerebellum	127	,,	
Pons	14·5	,,	
Medulla Oblongata	6	,,	
	960·5		

The main cerebral vessels to their third and fourth ramifications were in an exceedingly atheromatous condition, appearing in some places almost occluded by the thickening of their walls.

As regards the thoracic and abdominal viscera, it may be briefly stated that the lungs were emphysematous; the pericardium acutely inflamed with much effusion of lymph; the heart, weighing 18½ ounces, generally hypertrophied, especially the left ventricle; and that the kidneys each weighed 6½ ounces, had granular surfaces and mottled cortices.

Remarks.—This case is remarkable in the fact that permanent left hemiplegia—incomplete, it is true—was associated with a lesion in the right hemisphere, confined, as regards the motor area, to the paracentral lobule.

Paralysis, partial or complete, of the leg only, in connection with an affection of this lobule, would indeed be quite in accordance with pathological teaching; but the simultaneous implication of the arm is not altogether easy to understand, both central convolutions being intact, as regards their external aspects. The hemiplegia resembled the ordinary kind in the fact of the arm being more paralysed than the leg, nor was there anything—except it were the absence of facial paralysis—to suggest an origin for the affection other than the usual one of a lesion of the pyramidal tract.

The affection of speech would clearly come under the head of "amnesic aphasia," and the localisation of the lesion in the left temporo-sphenoidal lobe seems fairly to agree with that of other recorded cases.

The destruction of brain-tissue was doubtless in all parts due to thrombosis of the vessels supplying the areas in question.

Critical Digest.

RECENT VIEWS ON THE INNERVATION OF THE HEART.[1]

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Amongst the vital problems connected with the heart, some of the most prominent and important are: (*a*) The construction and mode of action of the mechanism by which its contractions are produced and regulated (its automatic muscular and *motor* nerve arrangements); (*b*) the sensorium is informed of its condition (its sensory nerve arrangements); (*c*) the nutritive condition of its muscular fibre is maintained and regulated (its trophic nerve supply); (*d*) the construction and nature of the nerve arrangements, by means of which it is brought into relationship with the other parts of the vascular system, and with the other organs of the body.

THE AUTOMATIC MECHANISM OF THE HEART.

The nervous mechanism of the heart is extremely complicated, and the manner in which it acts is far from being perfectly understood. It is a subject, however, of the greatest importance, and we must, therefore, consider it in some detail.

In the *first place*, it is necessary to remember that the contraction of the heart is automatic, *i.e.* it is due to impulses arising within the heart itself, and that, in conditions of health, the action of the two sides of the heart, *i.e.* of the two pumps, is synchronous. Under ordinary circumstances the heart beats in a perfectly rhythmical and regular manner, the number of contractions being, in the adult male, about 72 per minute.

Until quite recently it was supposed that the rhythmical action of the heart was entirely due to the periodical and orderly discharge of motor nerve force in the nerve ganglia, which are scattered through the organ; but recent physiological observations, more especially the brilliant researches of Gaskell, seem to show that the influence of the cardiac ganglia is not indispensable, and that the muscular fibre itself, in some of the lower animals at all events, possesses the power of rhythmical contraction.

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[1] Part of the first of a course of lectures on the Diseases of the Heart, delivered in the Edinburgh Extra-Academical School of Medicine.

Gaskell's observations seem to prove conclusively, that, in the tortoise, the automatic action of the heart does not depend upon any special rhythmical nervous apparatus, but that it is due to a property of rhythmical contraction inherent in the muscular tissue itself.

It is perhaps premature to conclude that the automatic contractions of the human heart are produced in exactly the same manner as the automatic contractions of the heart of the tortoise. We may, however, safely say, that in man, the rhythmical action of the heart must be due, either to the periodical and orderly discharge of motor nerve force from the ganglia which it contains, or to a rhythmical property possessed by the muscular tissue independently of any special nervous mechanism; and if we may judge by analogy, the latter view is possibly the correct one. And this we may term the *first step* in the comprehension of the mechanism of these complicated arrangements.

In the *second place*, it would appear that the stimulus to muscular contraction is the presence of blood, or rather the presence of blood under a certain pressure, in the cardiac cavities.[1] If the pressure is too low the stimulus is insufficient, and the muscle does not contract. If, on the other hand, the pressure is too great over-distension and a paralytic condition may result. Indeed the recently published experiments of Sewall and Donaldson seem to prove "that, within its working limits of internal pressure, the heart muscle has a remarkable power of accommodating the intensity of its discharges of energy, to the resistance to be overcome.'[2]

> "A fact continually forced upon our attention," say these writers, "was the great dependence of the systole, as to its energy and completeness upon the amount of fluid within the heart. Particularly was this noticed in regard to the thin walled sinus and auricles, when isolated in the manner to be described. Within tolerably low fluid pressures, the sinus and auricles become so distended as to be powerless to contract; as the pressure within them is reduced they contract feebly, and when the quantity of blood flowing into them is only just sufficient to bring about full distension during diastole, they contract powerfully, and empty themselves completely at each vent. As the supply of blood is further lessened by lowering the pressure flask, the contractions apparently again become feebler, and in the sinus hardly visible."[3]
>
> "The exact manner in which the cardiac muscle is stimulated

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[1] In some of the lower animals, the frog for instance, the heart continues to beat even after the cavities have been cleared of blood, and indeed when they are almost empty of fluid. In the frog, therefore, the presence of blood is not absolutely necessary to produce cardiac contractions, but it is nevertheless probable that the pressure of the blood in the cardiac cavities, under ordinary circumstances, acts as a stimulus, and excites the contraction of the cardiac muscular fibres.

[2] 'The Journal of Physiology,' vol. iii., p. 361.

[3] Loc. cit., p. 361.

by the presence of blood under a certain pressure is obscure. If the muscular contraction can occur independently of the ganglia, we must suppose either that the muscular fibre is *directly* stimulated by the mechanical stretching which it undergoes when the cardiac cavities become distended; or, that the contraction is indirect, and is due to stimulation of the fine nerve fibres[1] which, as Schweigger-Seidel[2] has shown, form a rich plexus in the endo-cardium. If we grant that the ganglia are concerned in the process, we may suppose, as some physiologists have for long held, that the mechanism is a reflex one. A cardiac ganglion-cell may be regarded as the centre of a reflex arc, to which a sensory nerve fibre passes from the endo-cardium, and from which a motor nerve fibre proceeds to the cardiac muscle (see fig. 1); the reflex mechanism being thrown into action by the presence of blood, under a certain pressure, in the cavities of the organ.

Fig. 1.

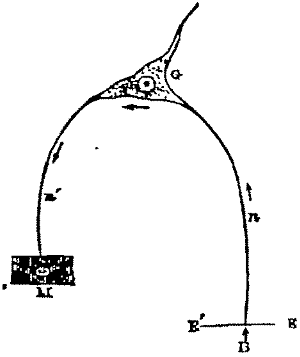

FIG. 1.—*Diagrammatic representation of the reflex mechanism by which (it has been theorised) the cardiac muscle (or muscular coat of the blood-vessels) is thrown into contraction, under the influence of the blood pressure.*

E′ E, the endo-cardium; B, the blood in the cardiac cavity; M, the muscular fibre of the heart; G, ganglion cell, the reflex centre; *n*, sensory nerve fibre conducting the impression generated by the blood pressure on the sensory nerve terminations under the endo-cardium to the reflex centre; *n′*, motor nerve fibril conducting the impulse from the reflex centre to the muscle.

In the case of blood-vessels, E E′ is the endothelium; the other references will be readily understood.

In the *third place*, the experiments of Gaskell, and of Sewall and Donaldson, seem to show that in the frog and tortoise at least, and when the heart is acting normally, the motor impulse originates in the sinus, and passes from the sinus to the muscular fibres of the auricle, and thence to the ventricle, the rhythm of the sinus determining that of the whole heart.

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[1] This view necessarily supposes that these fine nerve fibres are motor.

[2] Quoted by Power, Carpenter's 'Physiology,' p. 276.

Those authorities who believe that the automatic action of the heart is due to the periodical discharge of its motor ganglia, necessarily conclude, in order to explain these results, that the action of the ganglia, which are situated in the sinus venosus, is more powerful than that of the ganglia situated in other parts of the organ; and indeed some authorities have supposed that the ganglia of the sinus venosus are the true automatic centres, while the action of the other ganglia requires to be excited reflexly, or in some other manner. The supporters of the neurotic theory further believe that the sequence of the cardiac contractions (the fact that the contraction of the auricle follows at a regular interval upon the contraction of the sinus, and the contraction of the ventricle at a regular interval upon that of the auricle) is due to nervous influences.

Dr. Gaskell, whose experiments seem to show, as I have already mentioned, that the automatic rhythm originates in the muscular tissue itself, explains the fact that the contractions originate in the sinus, by the peculiar structure of the cardiac muscle in this part of the heart; while he states that the sequence of the contraction of the ventricle to the auricle is not due to separate stimuli passing along nerve fibres from the sinus to the ventricle—not, in short, to any nervous mechanism—but to the fact that a wave of contraction passes directly from the muscular fibres of the auricle to the muscular fibres of the ventricle, through the muscular fibres forming the auriculo-ventricular groove. The pause, or, more correctly, the alteration of rate in the progress of the contraction wave, which takes place between the contraction of the auricle and the contraction of the ventricle, is, he says, due to an alteration in the conducting power, which naturally exists at the auriculo-ventricular ring. "This diminished conducting power or natural block, exists not only because the auriculo-ventricular muscular ring is narrow, and that a somewhat abrupt change occurs in the direction of the muscular fibres along which the contraction wave passes when it reaches and leaves this ring, but essentially because the structure of the muscular fibres here is different from those of the auricle or ventricle.

"The muscle fibres throughout the heart[1] are," he states, "of the same type, any differences which are seen are differences in the prominence of the various structural peculiarities of the cardiac type of muscle, and are not so great as the differences between unstriated and striated muscle fibres. Thus all the muscle fibres are, to a greater or less extent, transversely striated; but the prominence of this striation varies considerably. Similarly the thickness of the fibre, the extent of parallelism of its edges, the size of the nucleus in relation to the size of the fibre, and therefore the extent of crowding of the nuclei in any strip, all present differences in different parts of the heart. The greatest contrast is seen when the muscular fibres of the ground layer in the sinus are teazed out and compared with the muscle fibres of the spongy tissue of the ventricle. The sinus muscle fibre is thin and delicate, tapering somewhat at both ends, with a large central oval nucleus which causes a distinct bulging of the fibre; the substance of the fibre shows a striation which is decidedly indistinct, presenting often a granular rather than a distinct banded appearance. On the other hand, the ventricular muscle fibre is boldly and strongly striated, it is much thicker than that of the sinus, its edges are parallel, and the thin elongated nucleus is small in comparison to the size of the fibre. The muscle fibres of the reticulated tissue of the auricle are not so large or so coarsely striated as those of the ventricle, though larger and much more distinctly striated than the sinus muscle fibres. Their edges also are more parallel than in the fibres of the sinus. The muscular ring forming the junction of the auricles and ventricle, and to a certain extent the whole junction wall joining the two auricles, are composed of muscle fibres, with a structure intermediate between the sinus and the auricle muscles. The nuclei are large, conspicuous on section both in size and number, the striation is not so well marked as in the bulged portion of the auricles, and the fibres are thin and delicate with somewhat parallel edges.

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[1] He is speaking, it must be remembered, of the adult heart of the tortoise.

"Such a structure as above described is very suggestive, not only as an explanation of the causes which occur naturally in the course of the peristaltic wave of contraction, but also of the differences of rhythmical power exhibited by different parts of the heart."[1]

Dr. Gaskell, therefore, conceives "that the variations in the rhythmical power and in conductivity, which are characteristic of the different parts of the adult heart of the tortoise, may all be accounted for on the supposition, that the development of the muscular tissue of the originally tubular heart has not proceeded at the same rate throughout the tube, so that in the adult heart greater variations in rhythmical power are apparent in the different sections of it, than in the original tubular heart; the peristaltic wave of contraction which originally passed smoothly from end to end, passes finally along a tube of irregular calibre, the muscular walls of which have become so modified in their rates of contraction and conduction, as well as in the arrangement of the fibres, as to form out of a simple peristaltically contracting tube such an efficient muscular pump as is represented by the adult heart.[2]

"The conception advanced above, that the rhythmical beating of the heart is due to a series of peristaltic contractions which start from that particular portion of the muscular tissue of the heart in which the property of automatic rhythm has been most largely developed, brings the heart's action into harmony with the rest of the vascular system and with the rhythmical properties which are so often manifested by the less specialized forms of muscular tissue."[3]

Space will not allow me to detail the facts and arguments on which Dr. Gaskell founds this theory, which obviously has most important practical bearings on the pathology of the human heart. I would, however, strongly advise my readers to peruse the original for themselves.

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[1] Loc. cit., pp. 72, 73.

[2] Loc. cit., p. 77.

[3] Loc. cit., p. 80.

In the *fourth place*, Gaskell has distinctly proved that the ventricle is supplied with afferent nerve fibres, which are "able to regulate the force of the auricular contractions, as well as in all probability the rate of rhythm. It is then conceivable that the function of many of the nerve fibres which pass into the ventricle is by their action upon the force of the auricular contractions to regulate the amount of blood thrown into the ventricle, and therefore the amount of work done by the heart."[1]

But although the movements of the heart are automatic, it is intimately connected both with the sympathetic and cerebro-spinal nervous centres; and its action can, as each one of us so well knows, be readily affected by general nervous influences. But in order that this most important and difficult part of our subject may, if possible, be clearly understood, I must now describe the nervous supply of the heart in some detail.

A description was here given of the nerves supplying the heart, and the subject was illustrated by means of a large coloured drawing, of which the wood-cut on p. 516 is a copy.

The Manner in which the Vagus and Sympathetic Nerves Affect the Heart.

I have now described the construction of the mechanism concerned in the innervation of the heart, and I must next point out the manner in which that mechanism works.

We have already seen that while the movements of the heart are automatic (i.e., are due to impulses arising within the heart itself), they can be profoundly modified by the condition of distant parts; and that the impulses which produce these modifications are conveyed to the heart through certain branches of the pneumogastric and sympathetic nerves. Now, until recently, we might have summed up the effects of these impulses by saying, that ***impressions passing to the heart through the pneumonogastric retard, while impressions passing to the heart through the sympathetic accelerate its movements*** (hence the terms "***inhibitor***" and "***accelerator***" of the heart which are given to the two nerves respectively); but recent observations seem to show that the action of the vagus on the heart is much more complicated, and that it affects not only the rhythm, but also the force of the cardiac contractions, and that it exerts a trophic influence upon the cardiac muscle. It will be necessary, therefore, to consider each of these functions separately.

The Inhibitory Action of the Vagus.

As the result of the laborious investigations of many able observers, physiologists have concluded:—*Firstly*, that there exists in the medulla oblongata a cardio-inhibitory centre, which is continually exerting a restraining influence upon the heart; and *Secondly*, that the action of this cardio-inhibitory centre may be intensified, i.e. the action of the heart may be still further retarded by:—

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[1] Loc. cit., p. 92.

(*a*) Direct stimulation, i.e. by certain changes in the medulla itself.

(*b*) Impressions passing to it from the brain.

(*c*) Impressions passing to it from peripheral parts (reflex stimulation).[1]

"Some authorities also believe that the cardio-inhibitory centre may itself be inhibited, i.e., its restraining power taken off the heart, by impressions passing to the medulla from other parts of the central nervous system, or from the periphery."

The most important local changes which stimulate the cardio-inhibitory centre, appear to be increased blood-pressure within the cranium, and irritative lesions in the medulla or in the neighbourhood of the vagi roots, e.g., inflammatory affections at the base of the brain, etc.; while reflex inhibition of the heart seems to be chiefly brought about by impressions passing to the medulla from the abdominal viscera.[2]

It has also been supposed that the action of the heart may be inhibited by impressions passing from the heart itself. Rutherford's experiments certainly seem to show that reflex inhibition of the heart can be produced in the rabbit by stimulation of the central cut end of the superior cardiac nerve, the impression passing up to the cardio-inhibitory centre in the medulla oblongata, and back, through the vagus and its inferior cardiac branch, to the heart.

"The ***superior cardiac branch*** arises by two roots, one connected with its superior laryngeal branch crosses down the neck close to the cervical spmpathetic, and joins one or two branches of the inferior cervical ganglion with which it proceeds to the heart. This branch is a purely sensitive nerve; and is in fact the sensitive nerve of the heart. On cutting it across, and stimulating its inferior (peripheral) end no effect is produced; while stimulation

[p. 518]

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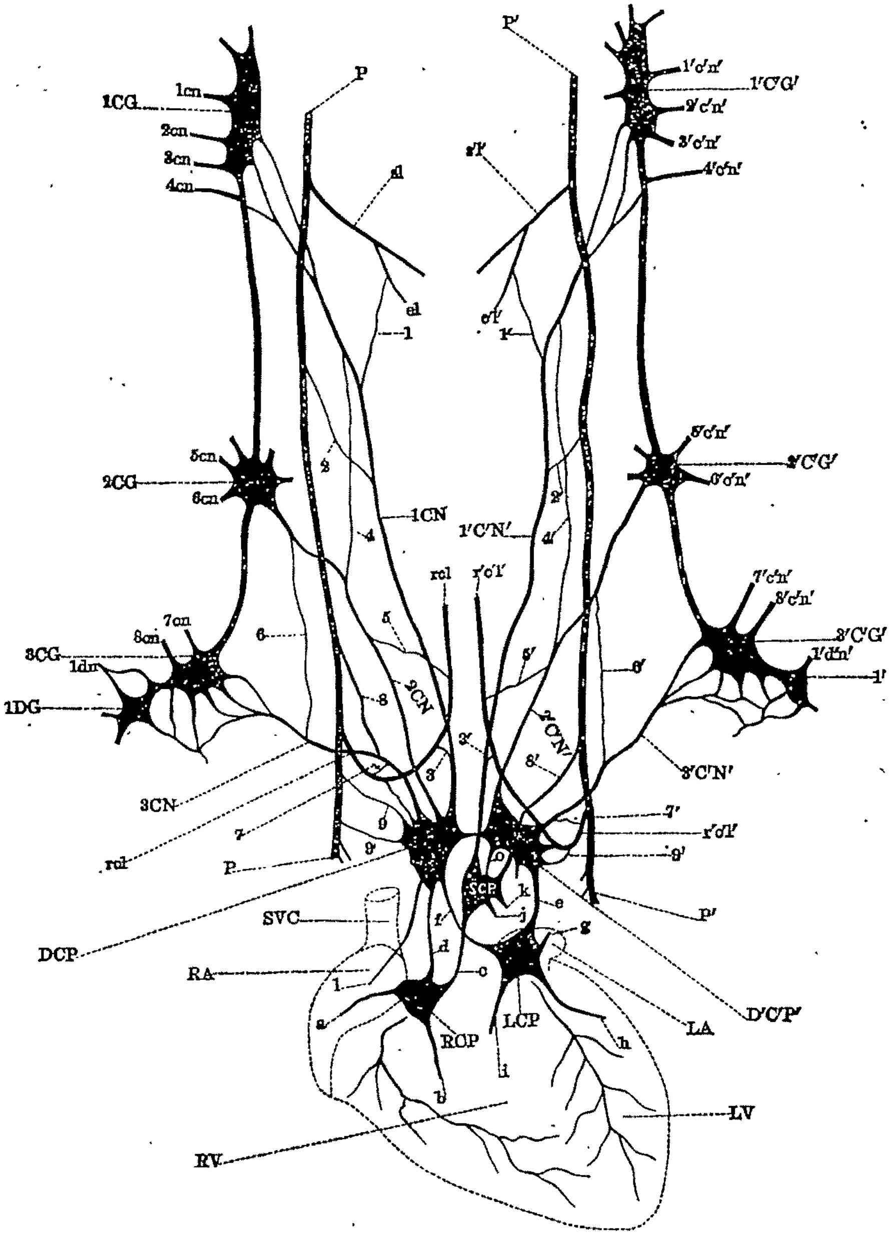
[1] Whether the action of the cardio-inhibitory centre is automatic or not, is still undecided.

[2] Powerful stimulation of any afferent or sensory nerve can probably produce reflex inhibition of the heart. Rutherford found that the cardio-inhibitory fibres of the vagus may be thrown into action by stimulating: (1.) The central end of the vaso-inhibitory or superior cardiac branch of the vagus (depressor nerve); (2.) The central end of the vagus of the opposite side; (3.) Almost any sensory nerve, in the case of warm-blooded animals; (4.) The abdominal viscera of the frog; (5.) The splanchnic and sympathetic.—'Lancet,' Dec. 16, 1871, p. 483. Michael Foster states that "the regulative action of the inhibitory mechanism is brought into more or less close connection with all parts of the body."—'A Text Book of Physiology,' p. 174.

FIG. 2.


P'
P
1'c'n'
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k
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e
P'
DCP
SVC
f
d
g
RA
1
c
a
LCP
RCP
LA
D'C'P'
h
i
b
LV
RV

Description of FIG. 2.—*Semi-diagrammatic representation of the nervous supply of the heart.*

RCP, right, and LCP, left coronary plexuses; SCP, superficial cardiac plexuses; DCP, right, and D′C′P′, left half of the deep cardiac plexus; P, right, and P′, left pneumogastric nerves; 1CG, right, and 1′C′G′, left superior ganglia of the sympathetic; 2CG, right, and 2′C′G′, left middle ganglia of the sympathetic; 3CG, right, and 3′C′G′, left inferior ganglia of the sympathetic; RV, right, and LV, left ventricles; RA, right, and LA, left auricles; SVC, superior vena cava.

1cn, 2cn, 3cn, 4cn, 5cn, 6cn, 7cn, 8cn, branches of the right, first, second, third, fourth, fifth, sixth, seventh, and eighth cervical nerves, proceeding to the sympathetic ganglia.

1′c′n′, 2′c′n′, 3′c′n′, 4′c′n′, 5′c′n′, 6′c′n′, 7′c′n′, 8′c′n′, branches of the left, first, second, third, fourth, fifth, sixth, seventh, and eighth cervical nerves, proceeding to the sympathetic ganglia.

1DG, right, and 1′D′G′, left first dorsal ganglia of the sympathetic; 1dn, branches of the right, and 1′d′n′, left first dorsal nerves proceeding to the first dorsal ganglia of the sympathetic.

SL, right, and S′L′, left superior laryngeal branches of the pneumogastric; EL, right, and E′L′, left external laryngeal nerves; rcl, right, and r′c′l′, left recurrent laryngeal nerves at lower part of neck, and in the thorax respectively.

a, branch from right coronary plexus to right auricle; b, branch from right coronary plexus to right ventricle; c, branch from the superficial cardiac plexus to the right coronary plexus; d, branch from the right half of the deep cardiac plexus to the right coronary plexus; e, branch from the left half of the deep cardiac plexus to the left coronary plexus; f, branch from the right half of the deep cardiac plexus to the left coronary plexus; g, branch from the left coronary plexus to the left auricle; h, branch from the left coronary plexus to the left ventricle; i, branch from the left coronary plexus to the right ventricle.

1CN, right, and 1′C′N′, left first or superficial cardiac nerves.

2CN, right, and 2′C′N′, left middle cardiac nerves.

3CN, right, and 3′C′N′, left inferior cardiac nerves.

1, branch from the right, and 1′, branch from the left external laryngeal nerves to the superior cardiac nerves; 2, branch from the right, and 2′, from the left pneumogastric nerves to the superior cardiac nerves; 3, communicating branch from the right, and 3′, from the left recurrent laryngeal nerves to the first cardiac nerves on the right and left sides respectively; 4, communicating branch between the right, and 4′, communicating branch between the left superior and middle cardiac nerves; 5, communicating branch between the right, and 5′, between the left middle cardiac and recurrent laryngeal nerves respectively; 6, communicating branch between the right, and 6′, communicating branch between the left, middle and inferior cardiac nerves; 7, communicating branch between the right, and 7′, between the left inferior cardiac nerves and the recurrent laryngeal respectively; 8, communicating branch between the right inferior cardiac nerves and the trunk of the pneumogastric, 8′, on the left side this branch of the pneumogastric proceeds directly to the superficial cardiac plexus; 9, 9, branches from the trunk of the right pneumogastric, and 9′, from the left recurrent laryngeal nerve to the right and left halves respectively of the deep cardiac plexus.

of its upper (central) end causes pain, retardation of the heart, and, as I shall afterwards more particularly point out, such a striking diminution of blood-pressure, that the term *depressor* nerve has been given to it.

"In the rabbit the vagus gives two branches to the heart, a superior and an inferior cardiac branch (see Fig. 3).

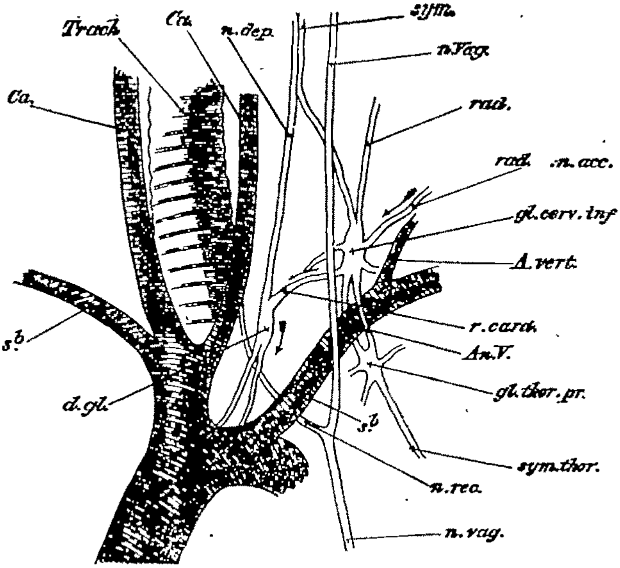

FIG. 3.—*The last cervical and first thoracic ganglia in the rabbit. (Left side.) (Somewhat diagrammatic, many of the various branches being omitted.) (After Foster.)*

Trach., Trachea. *Ca.*, carotid artery. *sb.*, subclavian artery. *n. Vag.*, the vagus trunk. *n. rec.*, the recurrent laryngeal. *sym.*, the cervical sympathetic nerve, ending in the inferior cervical ganglion, *gl. cerv. inf.* Two roots of the ganglion are shown, *rad.*, the lower of the two accompanying the vertebral artery, *A. vert.*, being the one generally possessing accelerator properties. *gl. thor. pr.*, the first thoracic ganglion. Its two branches communicating with the cervical ganglion surround the subclavian artery, forming the annulus of Vieussens. *sym. thor.*, the thoracic sympathetic chain. *n. dep.*, depressor nerve, which, though running by the side of the sympathetic, is really a branch of the vagus, from which it separates higher up. This is joined in its course by a branch from the lower cervical ganglion, there being a small ganglion at their junction, from which proceed nerves to form a plexus over the arch of the aorta. It is this branch from the lower cervical ganglion which possesses accelerator properties—hence the course of the accelerator fibres is indicated in the figure by the arrows.

"The *inferior cardiac branch* leaves the vagus below the origin of the inferior laryngeal nerve, and proceeds directly to the heart.

It is a purely motor nerve, and stimulation of its inferior cut-end gives rise to the same effects as stimulation of the vagus itself (i.e., stimulation above the origin of this branch), viz. (with weak currents) retardation of the heart from prolongation of the diastole, and (with stronger currents) complete arrest during diastole."

In the human subject, two branches of the vagus seem to correspond to the superior cardiac branch of the vagus in the rabbit; while the inferior cardiac branch of the vagus of the rabbit seems to be (mainly at all events) represented in man by the large branch which leaves the vagus trunk at the lower end of the neck (inferior cervical branch).

Now it has been supposed that these two nerves, together with the cardio-inhibitory centre in the medulla, constitute a reflex arc; and that stimulation of the peripheral terminations of the superior cardiac nerve in the heart may generate an impulse which, being conveyed to the cardio-inhibitory centre, and being reflected back to the heart along the vagus, may retard the action of the heart. Whether the superior cardiac nerve has any special function of this description, other than is possessed by all sensitive nerves, is extremely doubtful; and it seems certain that increased blood-pressure within the heart, as a whole, does not, as was at one time supposed, produce reflex inhibition of the organ.

Physiologists are not yet agreed as to the exact manner in which the vagus causes inhibition of the heart.

Until quite recently it was believed that while weak stimulation of the nerve prolonged the diastole, the duration of the ventricular systole being very little affected, more powerful stimulation caused complete arrest during diastole (complete relaxation), the next systole being as it were indefinitely postponed; and it was generally supposed that the vagus inhibited the contractions of the heart by preventing motor impulses being sent from the automatic ganglia to the muscle, i.e., by holding in, as it were, the motor energy of the ganglia; and this would appear to be the view which Sewall and Donaldson take. They think that their experiments "point pretty clearly to the conclusion, that the inhibitory action of the vagus nerve exerts itself in the same indirect manner (as a motor stimulus does), influencing the ventricle by damping in sinus and auricles the excito-motor discharges arising in these parts of the heart."[1]

Dr. Gaskell takes a different view. He has shown:—*Firstly*, that the vagus is able to modify all the great functional attributes of the cardiac muscle, viz.:—

(*a*.) The rate at which automatic contractions are produced. (The *automatic rhythm*.)

(*b*.) The force with which the contractions, more especially the contractions of the auricle, are carried out. (The *force of contraction*.)

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[1] Loc. cit., p. 367. 'Journal of Physiology,' iii.

(c.) The facility with which the contractions are conducted by the muscular fibres. (The *power of conduction*).[1]

Secondly, that it sometimes produces depression, at other times exaltation of function.

He believes that the vagus may produce standstill or inhibition of the heart (auricles and ventricles) in the three following ways:—

(1.) By producing cessation of its automatic contractions, i.e. by depressing the rhythmical power of the muscular fibres of the sinus in which the automatic rhythm originates. (This has been established in the frog, tortoise, and snake.)

(2.) By reducing the force of the auricular contractions, so that they become invisible, i.e. by depressing the contraction power of the rapidly contracting reticulated muscular fibres of the auricle. (Frog.) The contractions he states may be so small as only partly to be visible, so that to the naked eye the heart appears to stand still.

(3.) By blocking the contraction wave at the sino-auricular ring, i.e. by depressing the conducting power of the muscular fibres connecting the sinus and auricle. (Snake, tortoise, frog.)

The Trophic Function of the Vagus.

As I have previously remarked, Dr. Gaskell[2] has advanced the important theory that the vagus is the trophic nerve of the heart. "Although," he says, "the initial effect of the vagus is to depress some function, its final and most enduring power is to exalt, intensify, and repair that function. Thus, although it slows rhythm, yet its stimulation makes the rhythmical power last longer than it otherwise would, and makes the heart beat with regularity when it was previously irregular; although it reduces the force of the contractions, yet its ultimate effect is to improve and sustain the contraction force, although it may diminish the conduction power, yet in the end it completely repairs that power. *For these reasons*," he says, "*I look upon the vagus as essentially the trophic nerve of the heart.*"

Since this chapter was written I have learned from Dr. Gaskell that the term "trophic" as it is commonly used and understood does not exactly express the function which he supposes the vagus to have over the cardiac muscle. "I have felt," he says, "all along that the word trophic is somewhat misleading as applied to the results of my experiments on the action of the vagus. I wanted a word to express what appears to me to be the fact, in the cold-blooded animals at all events, that the inhibitory process is not destructive but constructive in its nature, but could not find any good term. I therefore used the term 'trophic,' not perhaps in its usual sense, but rather in a sense similar to that of Heidenhain when he divides the nerves supplying the submaxillary gland into secretory and trophic."

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[1] In the tortoise the force of the contractions of the ventricle was uninfluenced by vagus stimulation. In the frog, on the other hand, the vagus influences the force of the ventricular as well as of the auricular contractions.

[2] Loc. cit., p. 104.

The Mode of Action of the Vagus on the Heart.

The exact manner in which the terminal fibres of the vagus are brought into relation with the cardiac muscle is unknown. The effects produced by vagus stimulation, and by the action of certain poisons, have led some physiologists to believe that the arrangement is an extremely complicated one; and in order to explain these results, they have theorised that there existed an inhibitory mechanism within the heart itself, the action of which is strengthened (brought into play) by impressions carried to it by the terminal branches of the vagus, and which in its turn acts upon, i.e. inhibits the ganglionic apparatus more immediately concerned in the production of the rhythmical movements of the heart, i.e. the automatic nerve mechanism.

"The facts and arguments which they have advanced in support of the view that the vagus does not act directly upon the cardiac muscle, but that it acts through some intermediate nerve apparatus are as follows:—

"1. The latent period, which elapses between electrical stimulation of the vagus and the production of its inhibitory action on the heart is twenty times longer than that which elapses between the electrical stimulation of an ordinary motor nerve, and the contraction of its muscle, viz. $\frac{1}{5}$th and $\frac{1}{100}$th part of a second respectively.

"2. A very much stronger current is required to produce inhibition of the heart through the vagus, than is required to produce spasm of a voluntary muscle, when its motor nerve is stimulated.

"3. If the action of the heart be arrested by stimulation of the vagus, the rhythmical contractions after a time return, although the stimulation of the vagus be continually kept up, a fact which may be explained either by supposing that the inhibitory fibres of the vagus become gradually exhausted by the prolonged stimulation, or that the accumulation of nerve force in the automatic ganglia becomes after a time too great to be held back, and that an explosion and muscular contraction consequently take place.

"4. Continued stimulation of one vagus annuls or prejudices the action of the other.

"5. 'The effects of vagus inhibition are perhaps more marked when the electrodes are placed on the boundary line between the sinus venosus and the auricle, than over the vagus trunk itself.'—*Foster*.

"6. If during the complete relaxation which results from powerful stimulation of the vagus, the muscular tissue be mechanically irritated, it is still found to contract.

"While the existence of an intermediate (intra-cardiac) inhibitory mechanism between the terminal fibres of the vagus and the automatic nerve apparatus they think is proved by the following circumstances:—

"1. When the electrodes are placed on the boundary line between the sinus venosus and the auricles, more powerful inhibition is produced than when they are applied to the vagus itself.

"2. After the administration of urari, stimulation of the vagus no longer produces inhibition of the heart, but stimulation in the region of the sinus venosus will still do so.

"3. After the administration of atropia, neither stimulation of the vagus nor of the sinus produces any inhibitory effect. It is therefore concluded that atropia paralyses the intra-cardiac inhibitory apparatus itself.

"4. Muscarin and jaborandi in full doses produce exactly the same effect which is produced by powerful vagus stimulation, viz., complete arrest during diastole, an effect which is not prevented by the previous administration of urari, which, as we have seen paralyses the terminal fibres of the vagus, but does not touch the inter-cardiac inhibitory mechanism; but which is prevented by the previous administration of atròpia which paralyses the intra-cardiac inhibitory mechanism. Hence it is concluded that muscarin and jaborandi stimulate the intra-cardiac inhibitory mechanism."

Dr. Gaskell's view as to the mode of action of the vagus is different. He has shown that the action of the vagus on the cardiac muscle is in many respects identical with the action of a weak interrupted current. He believes that the nerve acts directly upon the muscular fibre itself, and he argues that the action of atropin, muscarin, and some of the other cardiac poisons to which I have referred above, can be consistently explained upon this view.[1]

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[1] "Both atropin and muscarin," he says, "produce the effects above mentioned, by directly modifying the various functions of the cardiac muscle, and in proportion to the stability of the effect so produced is the possibility of a modification of that effect by the direct stimulation of the muscle or by the action of the cardiac nerves. It is partly because the atropin acts on the muscular tissues in such a way as to keep the various muscular powers fixed in a relatively high state of activity, that the cardiac nerves are unable any longer to depress those powers. It is because atropin is able to restore the rhythmical power of the muscle, that stimulation of the vagus and of the sinus is no longer able to produce inhibition; it is because the atropized muscle contracts with the very first stimulus at its full strength, that neither the cardiac nerves nor the interrupted current is any longer able to diminish these contractions; it is because atropin repairs the conducting power that stimulation of the vagus is no longer able to prevent the contraction wave passing from the sinus into the auricles." Again, "atropin not only prevents the action of the vagus nerves by its direct influence on the cardiac muscle, but also by its action on the intra-cardiac nerves themselves, and perhaps especially on their ganglionic nerve cells. Muscarin, on the other hand, influences the muscular tissues, leaving the nervous action intact, at all events, until a very large dose

"Again," he says, "I have shown clearly that the vagus depresses and exalts all the different functions of all the different muscular tissues of the heart, whether the function in question is rhythm, contraction, conduction, tone, or excitability. Also, I have shown that depression of one function is not necessarily accompanied by a simultaneous depression of another function, and so also with the exaltation of function. Further the exaltation of each function is not necessarily dependent upon a previous depression; in each case the primary effect may, under certain circumstances, be exaltation and not depression. Combining these two facts together, we are driven to accept one of two alternatives, either the vagus contains a multiplicity of fibres, which can be divided into two groups after the fashion of Heidenhain, 1. Depressors (Hemmungs-fasern); 2. Augmentors (Verstärkungs-fasern); and further, each member of each of these groups has again its own special function, so that, for instance, the rhythm-inhibiting nerve fibre is different in kind from the contraction-depressing fibre, and so on; or else, the same trophic nerve fibre produces all the different effects observed, according to the nature of the muscle, which it supplies, and the condition of that muscle at the time.

"In this paper as well as in my previous paper, all the facts have tended strongly to prove that the vagus acts in the same manner upon the rhythm of the heart, and upon the force of its contractions, so that if separate inhibitory and accelerator nerve

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has been given. . . . Atropin then not only influences the muscular tissue, but also the nervous structures of the heart in such a way that neither the direct action of the interrupted current nor stimulation of the vagus nerves are any longer able to produce their usual effects upon the various functions of the cardiac muscles.

"The views put forward in this paper," he says, "necessitate a revision of the opinions at present held in respect to the action of a large number of cardiac poisons. The difference between the action of curare and atropin is one of the main arguments in favour of the existence of an inhibitory centre capable of being paralysed by atropin, but not by curare. Both poisons prevent the inhibitory action of the vagus nerve; but while after curare inhibition can still be caused by the direct stimulation of the venous sinus; after atropin this is no longer possible. The inhibition caused by muscarin can be removed by atropin, but not by curare. These facts do not, in my opinion, in the slightest degree prove the existence of an inhibitory centre; they simply confirm the well-known fact that curare is essentially a poison which affects nervous and not muscular stimulus, and give another proof of the affinity of the cardiac muscle and nerve to the ordinary striated muscle and nerve. Thus curare prevents the inhibitory action of the vagus, because it paralyses some part or other of the nerve endings. It does not prevent the inhibition caused by the direct stimulation of the venous sinus, because, unlike atropin, it does not act upon the muscular tissue of the sinus, and therefore cannot prevent the inhibitory action of the current applied directly to that muscle. For the same reason it is unable to influence the inhibitory action of a muscular poison like muscarin."—'Journal of Physiology,' vol. iv., No. 2, pp. 114–116.

fibres exist for the one, separate depressor and augmentator fibres must exist for the other. If, therefore, it can be shown that such diminution and augmentation of the rhythm of the contractions are due not to different influences reaching the muscle, but to the same influence affecting the muscle when its conditions are different, then it is, to say the least, highly probable that slowing and acceleration are also due, not to different nerve fibres, but to the action of the same nerve fibre under different circumstances. In the frog, as I have previously pointed out, such a complete gradation exists between a primary excessive diminution of the contractions and a primary augmentation in consequence of nerve stimulation, as to render the hypothesis that such curves depend upon the simultaneous stimulation of two antagonistic nerve fibres very improbable. I have now proved in addition, that a weak interrupted current applied to the smallest strip of cardiac muscle produces the same two opposite effects, and that here the depressing effect may be removed and the augmenting alone remain, when the condition of the muscle is altered by the application of atropin. In order, therefore, to still hold to the view of specific nerve fibres acting in opposite directions upon the force of the contractions, it is necessary to assume, not only that those nerve fibres possess opposite qualities up to their very termination in the muscle, but that even when the muscle itself is stimulated, the relative action of these two sets of nerves still holds its ground. In addition, atropin would have to be considered as acting upon these two sets of nerve endings, and not upon the muscle, paralysing the depressor nerve endings before those of the augmentor nerves. The experiments of Luchsinger and Szpilman, which lead them to the conclusion that atropin has a special action upon unstriped muscular fibre, those of Bowditch, already referred to, and my own, all point to the conclusion that atropin affects the cardiac muscle directly in consequence of its affinity to unstriped muscle fibre. The whole evidence goes to show that the vagus is a constructive and not a destructive nerve, that the initial depression of function is not of the nature of exhaustion, but is preliminary to a greater functional activity. The phenomenon presented by the muscular tissue of the frog and tortoise under the influence of an induced current, is of exactly the same nature as that seen in the muscle of the snail's heart (as pointed out by Foster and Dew-Smith) when a constant or weak interrupted current is sent through it. In both cases augmentation of function occurs as well as depression; in the one case the evidence is as strong in favour of special inhibitory and accelerator nerves as in the other. Clearly, in the case of the snail, it is simply impossible, as Foster has said, to explain such results by the presence of accelerator and inhibitory fibres in every strip of muscular tissue, when, as a matter of fact, cardiac nerves of any kind whatever have not been proved to exist. Clearly also, whatever explanation will ultimately be found for the action of the current upon the muscular tissue of the snail's heart, will also explain the same phenomena in the heart of the frog and tortoise. The problem which demands solution is essentially, Why is the condition of the muscle ultimately improved in every one of its functions by the direct application to it of a continuous stimulus, which is not strong enough to produce motor effects? and why is it that improvement of function is preceded in many cases by a diminution of function? When these two questions have received a satisfactory answer, it will no longer be strange that the vagus produces throughout two opposing effects, and the action of atropin will become clear; then the relationship between trophic and motor action will be understood, and the true functions of the ganglion cells found in connection with nerve fibres will be indicated. At present it can only be said that the vagus is the trophic nerve of the cardiac muscle, its action resembling that of a stimulus too weak to produce motor effects, and therefore it is possible that the function of the ganglion cells, in the course of the nerve, is to convert an otherwise motor into a trophic nerve."[1]

"This argument," Dr. Gaskell writes me, is based upon experiments upon cold blooded animals, how far it applies to the warm blooded I (Dr. Gaskell) do not yet know. Eichhorst and Tander conclude that the vagus in the warm blooded contains trophic fibres."

The action of the sympathetic upon the heart is even less clearly understood than the action of the pneumogastric, but it is believed that there exists in the medulla oblongata (or possibly above it), a centre, stimulation of which produces increased frequency of the heart's beat, and it is supposed that this accelerator, or cardiac-motor centre, as it is sometimes called, may be stimulated, i.e. the frequency of the heart's beat may be increased either by direct or reflex stimuli. The influences which throw this centre into action have not been very accurately ascertained, but it is probable that emotion excites the heart through the agency of the accelerator nerves; while diminished blood pressure within the cranium is supposed to stimulate them.

Impulses generated in this centre, the action of which, be it observed, is supposed to be intermittent (and in this respect to differ from the action of the cardio-inhibitory centre, which is supposed to be constant), appear to pass down the lateral column of the cord to the lower end of the cervical region, whence they pass through the nervi communicantes, to the (?) middle and inferior cervical ganglia of the sympathetic, and thence to the heart.

"According to Prof. Michael Foster the accelerator nerves frequently pass along the nerves accompanying the vertebral artery, and reach the heart through the last cervical and first dorsal ganglia."[1]

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[1] It may appear to some that I have entered into undue physiological detail in dealing with this subject, but it must be remembered that the mode of action of drugs is of the greatest interest in connection with the therapeutics of the heart. Further, I am fully convinced that it is only by attention to these minute physiological details that the physician can hope to make much advance in cardiac pathology, a subject on which our knowledge of the ordinary details of morbid anatomy is already so far advanced.

It is supposed from the long latent period which elapses between stimulation of the accelerator and the production of its specific action on the heart, that its fibres terminate in an intermediate ganglionic apparatus similar to that which I have already described in speaking of the termination of the pneumogastric.

Now, although the pneumogastric acts as inhibitor, and the sympathetic as accelerator, it must not be supposed that their action is directly antagonistic. That such is not the case seems abundantly proved by physiological observation. Physiologists have shown, says Dr. M. Foster, that "if during maximum stimulation of the accelerator nerves, the vagus be stimulated even with minimum currents, inhibition is produced with the same readiness as if the accelerator nerves were not acting. Vagus stimulation does not annul, but appears simply to suspend, during its continuance, the manifestation of the accelerator action."[2]

The Relationship between the Heart and the minute Blood-vessels.

In order to conclude the description of the innervation of the heart, I must shortly direct attention to the intimate relationship which exists between the heart on the one hand and the peripheral blood vessels on the other, and to the beautiful self-adjusting mechanism by which alterations in the one are of necessity followed by corresponding changes in the other.

We have already seen the fundamental importance of looking at the heart both as a mechanical pump and as a vital organ; and another point which I must now insist upon—and it is hardly of less importance whether we are studying the heart from a physiological, a pathological, or a clinical point of view,—is *the necessity of taking an all-round view*, so to speak, *of the circulation.* Indeed it is essential to remember that the heart is only a part of the vascular mechanism, and that the condition of the circulation, and consequently the condition of the heart (for the organ naturally adapts itself to the amount and kind of work which it has to perform) depend in no small degree upon the condition of the peripheral resistance, which is in its turn mainly due to the obstruction which the blood meets with, in passing through the minute arteries.

In conditions of health, the minute arteries are always more or less constricted in consequence of a permanent contraction of their middle or muscular coats, and it is to this 'tonic' contraction that the peripheral resistance is in great part due. [The peripheral resistance depends also upon the facility with which the blood passes through the capillary system of vessels; and since the blood flow through the capillaries may be influenced by alterations in (*a*) the conditions of the capillary walls, (*b*) the condition of the tissues outside the capillary walls, and (*c*) the composition of the blood itself, all of these factors must be taken into consideration as causes of peripheral resistance, and of variations in the blood pressure. In conditions of disease variations in the blood pressure may result from alterations in the "capillary resistance," but these variations are, in my opinion, never so great, either in conditions of health or disease, as those which are due to alterations in the condition of the muscular coat of the minute arteries.]

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[1] The middle and lower cervical ganglia in man appear to correspond to the lower cervical and first dorsal ganglia in the rabbit, through which accelerator impulses have been proved to pass, as shown in Fig. 2.

[2] 'Text Book of Physiology,' p. 175.

The degree of constriction is constantly undergoing variations even in health, and is subject to marked alterations in some diseased conditions; hence the amount of resistance which the heart has to overcome in forcing the blood into the capillaries, is constantly changing; and to meet sudden variations of this description a delicate self-adjusting nervous mechanism is provided. But in order that this part of our subject may be properly understood, I must now describe the mechanism by means of which the arterial "tone" is regulated.

There seems good reason to believe:—

(1) That the tonic constriction of the muscular coats of the minute arteries is (immediately) due to the presence of a peripheral neuro-muscular or (purely) muscular apparatus, which may be thrown into action by local stimuli, the most important of which is the blood pressure.

"The peripheral mechanism concerned in this maintenance of arterial 'tone' probably bears a close resemblance to the peripheral mechanism concerned in the production of the rhythmical movements of the heart.

"Ganglionic masses can be demonstrated in the walls of many of the minute blood-vessels, and it has been by some authorities supposed that the blood pressure produces stimulation of the muscular coats of these vessels in a reflex manner (see Fig. 17); but in many vessels (e.g. those in the skin and muscle) no such ganglionic masses have as yet been demonstrated, and in their case we must conclude that the contraction of their muscular coats is due to direct stimulation of the muscular fibre itself, or of the terminal vaso-motor filaments which are distributed to it.

"The view, which supposes that the muscular fibre is itself stimulated by the blood-pressure, is probably the correct one."

(2) That the action of this peripheral neuro-muscular or purely muscular mechanism is, under ordinary circumstances, maintained and regulated by impulses which are being *constantly* sent to it from centres (vaso-constrictor centres) in the medulla and spinal cord, through the vaso-motor nerves; and that the action of these vaso-constrictor centres may be increased or diminished both by direct stimulation or in a reflex manner. In other words, the calibre of the minute arteries, and therefore the peripheral

resistance, may be increased or diminished by central and peripheral causes (stimuli).

"The vaso-motor (i.e. vaso-constrictor) centre of the medulla, which is probably bilateral, is situated immediately above the calamus scriptorius, and extends for some distance into the pons (to 1 or 2 mm. below the corpora quadrigemina). The conducting fibres proceeding from it are believed to pass through the lateral columns of the spinal cord, and to proceed to the blood-vessels chiefly through the sympathetic system of nerves. The vaso-motor centre of the medulla probably regulates the vascular system of the abdomen, the arteries of the skin and muscles being probably mainly supplied by vaso-constrictor fibres proceeding from the vaso-motor centres in the spinal cord. The exact position of these spinal centres is not ascertained.

"The action of the vaso-motor (i.e. vaso-constrictor) centres, which it will be remembered is supposed to be constant, may probably be intensified by:—

"(*a*) Increased blood pressure within the cranium, and local irritative lesions of the medulla.

"(*b*) Impressions proceeding from the cerebrum.

"(*c*) Impressions proceeding from the periphery. It has been experimentally proved that irritation of any afferent (sensory) nerve, such as the sciatic, will produce constriction of the minute blood vessels (especially those of the abdomen) and increased blood pressure, presumably by reflexly stimulating the vaso-motor centre in the medulla."

(3) That the action of the peripheral neuro-muscular mechanism may be inhibited by nervous impulses passing to it from certain centres in the medulla, and (?) spinal cord, through the vaso-dilator nerves; and that the action of the vaso-dilator centres, which is not constant, may be called into play by central and reflex stimulation.

The calibre of the minute arteries may therefore be increased, and the blood-pressure lowered either by inhibition of the vaso-constrictor, or by stimulation of the vaso-dilator centres in the medulla and (?) spinal cord.[1]

"It is not absolutely certain that a distinct and separate vaso-dilator centre exists; some authorities in fact believe that the same centre may at one time evoke vaso-constrictor and at another vaso-dilator impulses; but it seems tolerably well ascertained that there are distinct fibres for the conduction of these two sets of impressions, and for theoretical and teaching purposes it is convenient to describe and represent a distinct vaso-dilator centre.

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[1] It must be remembered that the inhibitory action of the vaso-dilators on a peripheral neuro-muscular or muscular mechanism, is theoretical, and it is not definitely proved. "It is quite possible," as Foster says, "that dilation may be brought about in different ways, in different cases, and so also with constriction." —'A Text Book of Physiology,' p. 211.

"The exact course of the vaso-dilator conductors in the cord is yet undetermined, but it seems probable that they pass down in some part of the lateral columns, and reach the blood vessels chiefly through the cerebro-spinal (and not through the sympathetic) nerves.

"The vaso-dilator centre in the medulla is chiefly, if not exclusively, connected with the vessels of the abdominal viscera; and it is most readily thrown into action by stimuli passing to the medulla through the *depressor* nerve, and by impressions passing from the abdominal viscera through the splanchnics."[1]

Now, as I have previously stated, a very striking diminution of blood pressure results from stimulation of the superior cardiac branch of the vagus or *depressor nerve*, as it has therefore been called. It is obvious, therefore, that there is an important connection between the heart and the vaso-dilator centre in the medulla, by means of which the heart and peripheral blood vessels are brought into such close physiological connection, that changes at one end of the circulation, so to speak, are at once attended by compensatory changes at the other. If, for example, the arterial blood pressure is from any cause suddenly increased, a sudden strain is necessarily thrown upon the left ventricle, and it might easily happen that the resistance in front was too great for the force of the pump. Under such circumstances paralytic distention of the left ventricle and death would follow, if it were not for the fact that the undue distention of the cavity and the effort which the heart is making to overcome the resistance, produce irritation of the terminal fibres of its sensitive nerve (the superior cardiac or depressor nerve), in consequence of which a powerful stimulus is sent to the vaso-dilator centre in the medulla, with the result that a sudden dilatation, of the capacious blood-vessels of the abdomen, occurs, and that there is a rapid fall of the blood pressure. The peripheral resistance is in this manner immediately diminished, and paralytic distention of the left ventricle prevented.

It has likewise been thought that the vaso-dilator and vaso-constrictor centres are also connected with the cardio-inhibiting centre in the medulla; and some physiologists have supposed that reflex inhibition of the heart might be produced by the stimulation of the sensitive nerve of the heart (superior cardiac nerve), which presumably occurs in conditions of over-distention of the organ.

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[1] This description of the action of the vaso-motor mechanism is not complete. It must of course be remembered, that local variations in the blood supply are being constantly required by the ever-varying needs of particular times and organs, and that as Prof. M. Foster says, "the great use of the whole vaso-motor system is not to maintain a general arterial tone, but to modify according to the needs of the economy the condition of this or that vascular area" ('A Text Book of Physiology,' p. 213). But in studying cardiac affections the changes in the action of the heart, which are induced by local variations of this description, may, for practical purposes, be safely ignored. Hence I have limited my description to those alterations of the vaso-motor mechanism which are sufficiently general to produce definite changes in the action of the heart.

The recent experiments, however, of Ludwig and Luchsinger, and of Sewall and Donaldson, seem to prove that this is not the case, and that increased blood pressure within the heart, as a whole, generally weakens the inhibitory influence of the vagus. The last two observers, however, conclude "that changes of intracardiac pressure, *when experienced by the ventricle alone*" (no italics in the original), "are without effect on the cardio-inhibitory function of the vagus."[1] Possibly, therefore, when the ventricles only are over-distended, some reflex inhibition may occur; but even if it does occur, it certainly must be temporary and slight, for the main effect of stimulation of the sensitive nerve of the heart is reflex stimulation of the vaso-dilator centre, and consequent diminution of blood pressure throughout the body; and diminished blood pressure, as we have previously seen, tends to lessen rather than to increase, the inhibitory action of the vagus.

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[1] Journal of Physiology,' vol. iii., p. 363.

Reviews and Notices of Books.

Body and Will. By HENRY MAUDSLEY, M.D. Kegan Paul & Co., 1883. Demy 8vo., pp. 333.

OF all the faculties, qualities or divisions of mind, none is more obscure in nature, more difficult of investigation, or more imperfectly known than that of Will. The psychology of Cognition has been laboriously studied for ages, and our knowledge of it is the accumulated product of twenty centuries of investigation. If the current doctrine of cognition is not complete, it is at least very extensive, and he who cultivates this portion of the science must go again over ground that has been delved by innumerable predecessors. The psychology of Feeling is a younger and less matured science, but it has been prosecuted with vigour, and has reached a fairly advanced state of development. But our knowledge of Will is as yet rudimentary. It has scarcely yet reached a beginning. It is not yet settled what position should be assigned to Will among the other faculties of mind. It is even doubtful whether it should be looked on as a primitive faculty or no. The subject presents therefore to the original worker a great opportunity, and a task of great difficulty, both of which the author of this book appears in his preface to appreciate. "I am not ignorant," he says, "that those who are adepts in the schools of high philosophy may think the essay to be a weak intrusion into their high domains; for I must confess to being unable to use their language with a satisfactory sense of having clear and definite ideas beneath its terms, to having no proper faith in their methods, and to having failed to gather from their works fruits of any practical use." "I have no choice," he goes on to say, "but to leave the barren heights of speculation for the plains on which men live and move and have their being," and he speaks of "the necessity of taking serious account of facts and thoughts which, though they are not philosophy, may claim not to be ignored by philosophy." It appears therefore that Dr. Maudsley is fully alive to the shortcomings of his predecessors; that he approaches the subject from a new standpoint; that he works at it by new methods; that the aim of his book is to be before all things first intelligible, and then practical, and that theorising and speculation are no parts of his object. If we find upon examination that the book is both intelligible and practical, we may congratulate the author upon being successful as far as his main object is concerned, and may then go on to examine the validity of his doctrines, and the value of his method. If, on the other hand, we find him in the main either unintelligible or unpractical, we must conclude that he has failed in his primary objects, and we need carry the investigation no further. By this means the judgment arrived at will not depend on the opinion of an individual critic; but each reader of the criticism may form his own views of the merits of the book.

The book is divided into three Parts, of which the first treats of Will in its Metaphysical aspect; the second of Will in its Physiological, Social and Evolutional relations; and the third of its Pathological relations. The first part is mainly occupied with a refutation of the mediæval doctrine of free-will, a controversy that does not call for notice in a journal of neurology; but it contains also a chapter on the Physical Basis of Conscious Identity, which is the first chapter that professedly deals with the relation between body and mind, and which arouses therefore a heightened expectation. Proceeding to the study of the body with frank and open mind, Dr. Maudsley perceives that the essential principle of its being and function is a principle of individuation: that it is in fact unit and individual, an *ego*. This is plain and unmistakable on one point—that the body is an *ego*. A few pages further on the author says that "the *ego* is not a constant but a variable. It represents the aggregate of sensations clearly or obscurely felt at any given moment." Here the *ego* appears to be shifting its ground from the body to the mind. It still has extension, however, and "may it not actually be because the *ego* has extension that it can and does think space in every act of consciousness. . . .? Another consideration: Those who protest so much that the mind has not extension," &c. The metamorphosis is now complete. The *ego* which a few pages back was body is now mind. On p. 16, however, Dr. Maudsley says that "the *ego* is not any abstract entity, but the concrete individual . . . not any extremely sublimed and pure essence, but a feeling, acting, thinking organism." From this it appears that the *ego* is both body and mind, or at least that it is both the individual and the organism. And since things that are identical with the same thing are identical with one another, the individual and the organism are one and the same. But on p. 76 we read, "Are not in truth the individual's conscious memories of his affections and acts far less complete and actual, than the organisms registered memories of its affections and acts?" This sentence necessarily implies that the individual is not the organism, and the organism is not the individual. Thus Dr. Maudsley commits himself to the opinion that the individual and the organism are identical, and are not identical.

The modern doctrine of the parallelism and separateness of mental and cerebral changes is repeatedly accepted and indorsed by Dr. Maudsley; and he vigorously repudiates the supposition that a state of mind can enter into the circle of physical causation and produce an effect upon the bodily processes. On p. 120, he asks whether freedom of choice has, "as every other mental phenomenon confessedly has, a material equivalent in a particular brain action?" On p. 123 he says, "it will be wise not to describe the objective aspect of mental events in terms of the subjective, nor the subjective in terms of the objective indifferently, but to keep their respective languages apart." Again, p. 301: "Why not resolve to have a definite mental representation of the two invariably and essentially parallel processes when he [the pure psychologist] has to think of either? It would be an excellent check on vagueness of thought and expression, for it would help him to feel that he has a definite meaning in the abstract and somewhat empty psychological terms which he uses so freely, and to make others feel it, and would perhaps render his use of them a little more deliberate, exact, and sparing." Again, on p. 119 he speaks disapprovingly of "those who admit that physical and mental events go along together as exactly parallel phenomena, and who nevertheless bring the correspondence, so exact and constant up to a certain point, to an abrupt end by the arbitrary intervention of free will:" and he goes on to say (p. 120) "that to acknowledge the intervention of an ultra-physical factor in the chain of physical causation" would be "an implicit avowal that causation does not reign in human events, and that the science of human mind must always be metaphysical nescience." Thus there is no mental change without a physical change accompanying it, and to postulate the intervention of a mental factor as a disturbing influence in bodily processes is the negation of all science. On p. 109, however, Dr. Maudsley says that "the body has the power, in virtue simply of its physiological mechanism, without any help of will, to execute most complex purposive acts." This sentence seems to imply a possibility of will helping to execute acts; and in the next sentence this meaning becomes explicit. "The will has not the power to execute only, but it has the power to prevent execution, to hold impulses in check." The remainder of this paragraph speaks of the influence of the will over other mental states, and it is difficult, therefore, to know whether the words quoted mean a power of the will to act on the body; but in the following pages this uncertainty is removed. There the author mentions how "an act of will inhibits the movements of breathing." He explains that memory intervenes to check or inhibit a hasty movement. He thus acknowledges that intervention of a non-physical factor in the chain of physical causation, which, he says, is an implicit avowal that causation does not reign in human events.

How far Dr. Maudsley's endeavour to be practical has succeeded, is for the reader to judge, bearing in mind that practice cannot be founded upon contradictions, and that the questions hitherto dealt with are the most important and fundamental that are connected with the relation between Body and Mind. Even more earnest than Dr. Maudsley's desire to be practical, however, is his desire to be intelligible. He insists so often and so strongly upon the importance of this quality in psychological discussions, that it may be said to be beyond question the chief aim of his book; and if he succeeds in making himself generally intelligible, he may still claim to have scored a success. Some of the expressions indicative of Dr. Maudsley's desire for accuracy of thought and distinctness of expression have already been quoted, but the prominent place which he assigns to these desiderata as objects to be striven for, appears more from the frequency even than from the earnestness with which he speaks of them. Again and again at intervals of a few pages he urges throughout the book the importance of having clear ideas, and of expressing them clearly. His preface has been already quoted. On p. 5, he cautions us that "many persons do not thoroughly consider whether they distinctly know their own meaning, but deceive themselves in imagining that they have any distinct meaning at all." On p. 15, he says, "it will be well to examine rigorously," "it appears, when we consider the matter closely." On p. 36 he advises us to "let the inquiry be sincere and searching." On p. 69 he says that "it were much to be wished that the philosophers of the study would consider frankly and loyally." On p. 75 he proceeds to the study of the body "with a frank and open mind." On p. 78 he notices how, "recoiling from the danger of intruding upon sacred ground, and from the hardly less deterrent difficulty of resolutely forming clear and definite ideas and expressing them in exact terms and phrases, men have persistently dealt with words instead of things, and with words as things. On p. 84 he recommends "a frank and searching inquiry." On p. 149 he says that "to realise the full meaning of psychological facts, to get clear and exact notions of them in their mental relations is a difficult business, and one which those who have psychology as the method of introspection seem unable to accomplish." These are but a tithe of the instances in which Dr. Maudsley expresses his resolution to form his judgments with the utmost care, and to express them in the plainest terms. If he has succeeded in doing this, he has achieved the main object of his book. Let us take, therefore, a few sentences from the important parts of the book, and examine them with a frank and open mind, to see if we can gather from them clear and exact notions of mental and bodily relations.

The doctrine of the nature of the *ego* has, on account of its importance, already been quoted, and for the same reason it may now be referred to again. Speaking of the sensations which the *ego* represents, the author says (p. 80), "these sensations themselves representing the sum of multitudes of activities that are going on below the threshold of consciousness, and which, albeit unperceived and unfelt immediately, vibrate subtilely (*sic*) in the most intimate and intricate interactions of organic depths, and in the result affect deeply the *tone* of consciousness." It is not certain from the context whether it is the sensations or the activities that vibrate, but let us follow Dr. Maudsley's directions and try to realise the vibration of a sensation or an activity. Having got a definite notion of that, let us add the quality of subtileness, and imagine them vibrating subtilely. Now go a step further, and imagine this vibration occurring in an interaction. Again, imagine an organic depth; and yet again, get a firm grasp of the notion of the interaction of organic depths. Now combine all these notions together, and imagine an activity or a sensation vibrating subtilely in the interactions of organic depths. If the reader finds in this expression exact terms and phrases, and can gather from it clear and definite ideas, Dr. Maudsley has succeeded in his object.

Take another most important extract—a declaration of the nature of Mind. "It is," says Dr. Maudsley (p. 128), "*continuous* and *dependent*; for it is a becoming from the basis of all human past through the means of an essential co-operation of surroundings." Let us consider the matter closely and get a clear idea of the basis of all human past; then imagine a becoming from it; then picture to ourselves a co-operation of surroundings; make this co-operation essential, and finally imagine this becoming as happening through the means of this essential co-operation of surroundings. Let the reader follow Dr. Maudsley's instructions; let him make his inquiry sincere and searching; let him not deceive himself into the belief that he finds in these expressions a meaning, if they have no distinct meaning at all; and if he is successful in finding a clear and definite meaning, Dr. Maudsley has succeeded in the main object of his book.

In matters so abstruse as the nature of mind or the physical basis of conscious identity, it is perhaps impossible for an author to make his meaning quite clear, however great may be his desire to do so; let us therefore take some simpler subject, such as the origin of the social habits of ants, and see how the author expresses his meaning in this case. "We may suppose," he says, "that the societies of ants have survived as lessons of what might have taken place in other animals under more favourable auspices, and that their social union became an actuality of organic development, instead of being arrested as a possibility only, by reason of their burrowing habits in the construction of their habitations, of the smallness of their ingeniously constructed bodies, and of their tenacious industry, of their prolific natures, of their numbers and of the strength of their social union." The sentence is clearly expressed; but if we take it in the ordinary meaning of the words, it will appear that the social union of ants became an actuality of organic development by reason, amongst other things, of the strength of their social union; which can scarcely be the author's meaning.

Where the subject-matter is less abstruse, the author is more easily intelligible. He has a chapter on Evolution, to which he adds another process called Involution, of which latter he says that it is easier to entertain the general notion of a process of involution than to put forth an intelligible exposition of it. But he impresses on his readers that they ought "to recognise, investigate, and describe three processes, namely, (*a*) Involution, (*b*) Evolution, and (*c*) Dissolution; which processes, though three when viewed in relation to individual parts, are not three, but three aspects of one process, when viewed in relation to the embracing whole.

"Two leading facts, then, and for us ultimate facts, which it behoves us to apprehend and fix firmly in mind, are—first, that there has been what we may call a nisus of evolution in nature; and secondly, that progressive transpeciations of matter have been events of it. Continuity of nature, certainly, but as certainly not of kind in nature; for the continuity is of different kinds, therefore in some sort a discontinuity, a new kind springing from the basis of the old kind; not continuity by homogeneous, but by heterogeneous generation." No one, I venture to say, will have the least difficulty in clearly apprehending this doctrine who has thoroughly mastered the Athanasian creed.

How far Dr. Maudsley has achieved his aim of resolutely forming clear and definite ideas, and expressing them in exact terms and phrases; how far he has avoided the danger which he points out of dealing with words instead of things, and with words as things; how far he has a definite meaning in the abstract terms that he uses, and makes others feel it, each reader must decide for himself. The foregoing extracts will give the reader ample opportunity of judging of the style and form of Dr. Maudsley's utterances. We may now briefly consider the value of the doctrines that are so presented. "Having," he says, on p. 105, "now done so much to clear the ground, and to set the problem in its true light," &c. It is natural to turn back from such a sentence and see what it is that Dr. Maudsley has done to clear the ground, and to set the matter in its true light. What he has done is sufficiently remarkable. He has propounded a new theory of Mind. We have already learnt that mind is a becoming from the basis of all human past; but this is not the point of view from which it is regarded in the theory now under consideration. The passage in which the theory is set forth is too important not to be quoted entire. It begins as follows:—"Is it not the fact, indeed, that the undulatory theory of light was first suggested by the undulations of sound? In like manner the gulf between the conception of the movements of the cerebral molecules and the self-consciousness of will-energy may well be due to different ways of acquiring them; molecular motion and will be one and the same event, seen under different aspects, and to be known as such one day from a higher plane of knowledge." There is a scarcity of verbs in this sentence, but the meaning is fairly clear. Will is the subjective aspect of what on the objective aspect is molecular movement. The author goes on to say: "Perhaps when that time comes, the theory of an all-pervading mentiferous ether may help to bridge over the difficulty. For if the object and the brain are alike pervaded by such a hyper-subtile (*sic*) ether; and if the impressions which the particular object makes upon the mind be then a sort of pattern of the mentiferous undulations as they are stirred and conditioned within it by its particular form and properties; and if the mind in turn be the mentiferous undulations as conditioned by the convoluted form and the exceedingly complicated and delicate structure of the brain; then it is plain that we have eluded the impassable difficulty of conceiving the action of mind upon matter

—the material upon the immaterial—which results from the notion of their entirely different natures. Here, in fact, is a theory that gets rid at the same time of the gross materiality of matter and of the intangible spiritualities of mind, and instead of binding them together in an abhorred and unnatural union of opposites, unites them in a happy and congenial marriage in an intermediate region, and, if I may so speak, in an intermediate substance; a substance which, mediator-like, partakes of the nature of both without being exclusively either."

From this it appears that the difficulty of conceiving mind in terms of matter disappears if only we imagine the particles of matter as very small. According to Helmholtz, the smallest particle of matter that can ever be distinctly defined under the most powerful microscope is about 1-80,000th of an inch in diameter. Supposing this particle to be spherical, and to consist of water, it would contain, according to Mr. Sorby, 8,000,000,000 of molecules, each molecule consisting, as we know, of three atoms. Notwithstanding the amazing degree of tenuity that this supposition ascribes to the particles of matter, the division between the operations of mind and the undulations of the molecules of water remains absolute; so absolute that a great thinker has said that "we do not possess the intellectual organ, nor apparently any rudiment of the organ which would enable us to pass by a process of reasoning from the one phenomenon to the other." It is for the reader to judge whether the transition will be rendered more easily conceivable, by imagining a still greater attenuation of the particles of matter.

If the mind be itself made up of mentiferous undulations, can these undulations be stirred within the mind by its particular form and properties? and can the reader conceive, by help of this theory, a substance which partakes of the nature of both mind and matter without being exclusively either? A few analogous but much less difficult cases will prepare his mind for the task, and afford him good practice before attempting the major problem. Let him imagine a substance which is partly a triangle and partly a smell, or one which partakes of the nature of both swiftness and sweetness without being exclusively either. If the reader is able by means of the theory of a mentiferous ether to rise from these comparatively simple and concrete conceptions to the much higher conception of the blending of mind with matter, then Dr. Maudsley has achieved the greatest success in mental philosophy of modern times; if not, this theory of mind is a failure.

It will not be necessary to follow Dr. Maudsley's treatment of the various subjects considered in his book. They are sufficiently numerous, varied, and important, to occupy even a larger volume, including, among many others, the question whether animals have souls, the invention of lucifer-matches, the rise of Christianity, the Exodus, the fall of man, the origin of dermoid cysts, the morality of science, the doctrine of the Atonement, the habits of ants and the possible nature of their religion, the immortality of the soul, the failure of the Christian morality, the belief in God and the existence of the Devil. It is a book *de omnibus rebus et quibusdam aliis.*

It would be presumptuous in me to offer an opinion upon a book of Dr. Maudsley's, and I have not ventured to make any critical observations. In the foregoing notice the reader will find a sufficient account of the book to enable him to form his own judgment upon it, and he will find in addition certain principles laid down upon which he may, I think, safely and properly rely in estimating its value. The issues are largely questions of fact, and the verdict must be left to the voice of the public.

CHARLES MERCIER.

Les Maladies de la Mémoire. Par TH. RIBOT, Directeur de la Revue Philosophique. Deuxième édition. Paris, 1883. 18mo. pp. 168.

To the obligations that psychologists already owe to him, M. Ribot has added another by the production of this charming work, which is written with all the lucidity characteristic of his nation, and with the candour and modesty, as well as the happy union of precise scientific form, with attractive and interesting matter, which his previous works have led us to expect. The work does not contain any new principle; it is a systematic application of established principles to a class of phenomena to which they have not hitherto been so applied, with the result of organising an additional province of our knowledge. The keynote of the book is that memory is the subjective side of a nervous process, and that disorder of memory is the subjective expression of disorder of this process; and the main object of the author is to determine the nature of the disorder that affects the nervous process in each disorder of the memory. In some cases he succeeds in definitely indicating the nature of this disorder; in other cases he does not; and his great merit is that he never attempts to stretch his principles beyond their legitimate tether, or to deceive either his reader or himself as to the success of his efforts. He does what he can; and when he meets with a case that is inexplicable he candidly says so, and does not attempt to hide his ill success under a cloud of meaningless expressions.

The first third of the book is occupied with the consideration of the normal memory, and here M. Ribot lays down a wide and secure basis for the remainder of his subject. Conscious memory, he says, includes three things: the conservation of certain states of mind, their reproduction, and their localisation in the past. But conscious memory or remembrance is but a part, and a small part, of memory. Every case in which a new state is produced in the organism, and is preserved and reproduced, is a form of memory; and the vast majority of such cases have no conscious accompaniment. While every conscious memory has a biological substratum—implies a physical change in the organism—there are abundance of changes in every respect similar, save that they are not attended by consciousness. Memory is in fact essentially a biological process, and only accidentally and exceptionally psychological. Every case in which a set of movements is acquired, is a case of organic memory. Walking, writing, handicrafts, games of skill, are acquired in precisely the same way, and their acquisition is subject to the same influences as the words of a play or the lesson of a scholar. The two sets of memories resemble one another in every particular save in the presence of consciousness. Nay, they are not materially different even in this respect; for the first are attended by a vivid consciousness, until they are so completely acquired as to have become automatic; while the second, by constant repetition, may cease to have any accompaniment of consciousness. It is manifest that the two memories are essentially the same, and that the same organic process underlies them both. As to the nature of this process, M. Ribot accepts without modification the prevalent modern doctrine. The performance of an act, and the occurrence of a thought, are alike conditioned by the occurrence of a certain process in the nervous tissue; when a particular process takes place in nervous tissue, it leaves in the tissue a permanent modification of such a nature that, if any part of the process is again set going, there is a tendency for the whole process to be repeated. It is scarcely necessary to say that the doctrine that a single remembrance is imprinted on, or stored up in, a single cell is rejected by M. Ribot; but although he deprives the nerve-cells of this part of their prestige, he still attributes to them, it appears to me, a greater share in the process than can fairly be claimed for them. The nerve-cells are the tyrants of the physiological world. Not only can no bodily process be supposed to go on without them, but it is assumed and taught, not only by M. Ribot, but I think universally, that they and their prolongations are the only active portions of the nerve centres. The cell is the dynamite cartridge, and the rest of the grey matter is only the cotton-wool that it is packed in. M. Ribot admits the cell and its prolongations only to a share in the physiological process of memory. He shows that the number of cells—six hundred millions, according to Meynert—is numerically sufficient. He compares them to the letters of the alphabet, of which only twenty-five can be combined to form millions of words. And he infers that the possible number of combinations of the nerve-cells is great enough to satisfy the demands of the memory of the most learned of men. But of the intercellular substance he says not a word. It is time to renew the rebellion set up by the late G. H. Lewes against this despotism of the cell. What right has it to a position of functional supremacy? I know of none. If cells formed the bulk of the grey matter, as they form the bulk of the liver, and if the intercellular substance were present in insignificant amount, then the position might reasonably be conceded to them; but this is not so. When a section of the grey matter from any region of the nervous system is examined under a low power, the area occupied by the cells (neglecting those of "granular layers") does not preponderate over the area occupied by the intercellular space; on the contrary, the intercellular substance always preponderates, very often preponderates largely, and often is the only element in the field. If the nerve-cell were a constant constituent of grey matter, it might be conceded that the cells were necessary to the occurrence of a nervous process, although it might well be denied that they took the leading part. But their presence is not constant. Not only are there whole columns of grey matter in the spinal cord of man, with few or no cells in them, but in some fishes there are whole lengths of the cord without any cells at all; and in others the whole cord contains only a single cell here and there. The worship of the cell appears to have had its origin in a false analogy drawn between the structure of the grey matter and the structure of the other solid viscera. In the liver, for instance, and in the kidney, the cells are undoubtedly the active element, and the remaining elements have little other function than that of supporting the cells and binding them together. The superficial structural similarity of the brain to these organs appears to have led to the assumption of a functional similarity. Since in the one case there are cells and intercellular material, of which the former are active and the latter inert, and since in the latter there are cells and intercellular material; therefore in the grey matter also the cells are active and the intercellular material inert. The analogy is fallacious and the conclusion vicious. The intercellular material of the liver and the kidney is known to be of a totally different nature from that of the cells; developed from a different layer of the blastoderm, and possessing no community of constitution or property. A large part of the intercellular material of the grey matter is known to be identical in nature with that of the cell, and the remainder is not known to be different from it; it is developed from the same layer of the blastoderm, and the only valid inference with regard to its properties is that they are similar to those of the cell. The processes of the cell are prolongations of its own substance, and are undistinguishable from it in structure. They ramify into a network of fibres, which occupies a great part of the space between the cells. The substance of these fibres is thus continuous with that of the cells, and if we make any assumption about their properties, we must assume that the properties of the fibres are similar to, if not identical with, those of the cell. The remainder of the intercellular space is occupied by the neuroglia, and nothing could be more significant than the fact that while all observers speak of differences between the neuroglia and the nerve tissue, no absolute distinction has ever yet been drawn between them; and the best observers speak doubtfully and differ among themselves as to which cells and fibres should be ascribed to the neuroglia, and which shall be considered purely nervous. There are many excellent reasons why the intercellular grey matter should be looked on as taking an active part in the nervous process that constitutes the physical basis of memory, but these may be now disregarded. Seeing that the cells form but a small portion of the nerve centre, seeing that the other and larger portion is in part known to be closely similar in substance to the cells, and in the remaining part not known to be different from them, the onus of proof lies upon those who contend that the nerve-cells with their traceable prolongations are alone directly concerned in the function of the grey matter, and proof of this is impossible. The hypothesis is narrow and unreasonable; it rests upon no sound basis; and presumption is entirely against it. The cell-despot, like many another usurper, reigns neither by inherent right nor by the power of his active supporters, but by virtue of a general indifference, and because no one disputes his title. Let us repudiate this *cytolatry*.

Defects of memory are divided by M. Ribot into those which are limited to one class of acquirements, and those which affect simultaneously all the forms of memory. Of the former class aphasia is the type, and of the latter the loss of memory that occurs in old age is an example. General amnesias are again divided into the temporary, such as those which occasionally follow a blow on the head; the periodical, including cases of so-called "double consciousness;" the progressive, which are the commonest, the least curious, and the most instructive; and the congenital. Of each form a few examples are adduced, which have for the most part already done good service in the literature of the subject, and reasoning upon these, M. Ribot goes on to enunciate two important conclusions. The first of these has reference to the curious phenomenon of double consciousness, for which an intelligible physical basis is suggested; and the second is the Law of Regression. The division of consciousness that is found in cases of periodical amnesia is ascribed to an alternating condition of cœnæsthesis. Although the cases are rare, they are so extraordinary that they have attracted much attention, and their facts are tolerably familiar. After some critical event—usually a prolonged sleep—a person awakes with a complete forgetfulness of her whole past life, perhaps with a great change of character. She lives for some time in this New state, and then after another sleep she wakes with a normal memory of all the experiences of her life before her first sleep, and an oblivion of all that had happened in the New state. Another sleep reverses the condition of things. She now remembers the occurrences of the former New state, but is oblivious of everything else; and the Old state and the New state may alternate an indefinite number of times, the acquisitions of each holding good only for the periods of its own recurrence. To find a physical basis for this alternation, M. Ribot investigates first that of the conscious *ego*, a rock upon which many a psychologist has run hopelessly aground, but which affords M. Ribot opportunity for a display of dexterous navigation. The conscious *ego*, considered as it exists at any one moment, is the sum of all the states of consciousness then existing; of these states memories form a large part; and it is the sum of these memories which forms the consciousness of continuous identity. The number of different memories that enter into any state of consciousness is very great, and the group so formed is in a state of continual flux

—some memories continually falling out of consciousness and others rising into prominence. In this scene of perpetual change there is, however, one thing that persists. This is the cœnæsthesis—the obscure consciousness which is the result of all the vital actions, and which constitutes the knowledge of our own bodies. It is to changes in the processes that underlie this part of consciousness that M. Ribot attributes the breaks that sometimes occur in our consciousness of continuous identity; and it is to changes in these processes that he would attribute not only cases of periodical amnesia, but also those cases of insane people, who imagine that their bodies are changed into glass or wood, or that their sex is changed, or that they are dead. As to what the precise bodily change is that underlies the change in the cœnæsthesis, or how it is brought about, M. Ribot wisely refrains from guessing.

The Law of Retrogression we are already familiar with, under the name of the Law of Dissolution. The rule is that those requisitions are first lost which are most recent, least often repeated, feebly associated with others—in a word, which are least securely fixed in the nervous organisation. Those memories endure longest which by early acquisition, by frequent repetition and long duration, have been most completely "organically registered."

The consideration of partial amnesias is prefaced by a useful protest against the method of viewing memory as a single integral faculty. There is in reality no such thing as *the* memory; what we experience are *memories*, and in studying the subject this should be always borne in mind. Virtually the study of partial amnesias is limited to the loss of signs (spoken and written words, interjections and gestures); and this is followed by M. Ribot on familiar lines.

A brief consideration of cases of exaltation of the memory leads to the question—Do we ever forget? and this receives the answer that is probably nearest the truth. "It is possible," says M. Ribot, "that certain modifications of cells [I should prefer to say of nervous tissue], and certain dynamical associations are too unstable to endure. We may say, however, that persistence is, if not the invariable rule, at least the rule; that it holds good in the immense majority of cases."

In his conclusion the author shows that while the conservation or organic registration of a memory is an affair of nutrition of the grey matter, its reproduction depends on the state of the circulation therein.

The account here given will be enough to show that M. Ribot has produced a very valuable work, but it cannot convey the charm of the author's style, nor can it hope to emulate the clearness and precision of language which makes a most complex and difficult subject appear childishly easy. Much, in fact most, of what M. Ribot says, has been said before, but it has never before been said so clearly and unmistakably, nor in so convincing a manner.

CHARLES MERCIER.

Traité des Névroses. Par A. AXENFELD. Deuxième édition, augmentée de 700 pages, par HENRI HUCHARD; pp. 1195. Librairie Germer Baillière et Cie., Paris, 1883.

IN 1863 Axenfeld published the first edition of this work. While preparing the second, he in 1876 succumbed to a mortal illness. Anticipating the fatal termination of his malady he confided the conclusion of his literary undertaking to Dr. Huchard, who, during the year 1883, has produced the volume before us. This practically consists of the combined labours of both authors, as the original work of Axenfeld, consisting of 520 pages, remains essentially as before, but has been augmented to the extent of nearly 700 pages by M. Huchard, whose share can always be recognised by its being placed between parentheses. The volume as a whole, while professing to retain the general plan and originality of Axenfeld, has been brought up to the level of scientific knowledge at the present date, and all the modifications and improvements of the last twenty years have been added by the Editor.

Of an undertaking of this size and importance it is impossible at the present time to give a minute critical digest, it is sufficient to say that the work consists of a series of memoirs on the different neuroses, each of which is in itself complete, and embraces in a brief yet comprehensive manner all that is known of the subject. To every section is attached a most voluminous bibliography, the compilation of which represents an enormous amount of research and labour, and which supplies most valuable and extensive material for reference. The text is clear, concise, and well arranged, and from its completeness and general excellence, the work may be looked upon as a standard one, and well worthy to be placed on the shelves of every physician. Having thus summed up the merits of the volume, it is not necessary to further dilate on its details; but there are some general questions with reference to the subject of which it treats, which may be discussed with interest.

In the first place it may be asked, What is a neurosis? Axenfeld defines the term as follows. "A morbid state, most usually apyrexic, in which there exists an exclusive or at least predominant modification of the intelligence, sensibility or motility, or all these faculties combined; a morbid state in which there is this double peculiarity, that it may be produced in the absence of any appreciable lesion, and does not by itself induce profound or persistent structural changes." Like most other definitions of natural processes, this is not a perfect one, and even its author admits that it is not complete, but only approximative. At the same time, it as nearly as possible expresses the position of a very complicated state, and one very difficult to differentiate in words. The term "neurosis," although of British origin—Cullen having been the first to introduce the word—is less employed in this country than it is abroad, the expression "functional nervous disease" having been substituted for it. There can be little doubt, however, that the former is the preferable terminology, and Neurosis, a word involving little theory, is a convenient term to express that class of affections, to which the appellation of functional has added confusion and given rise to much controversy.

Next arises the question, Is there a place in medical nosology for these neuroses or so-called functional nervous disorders? In practice, two classes of disease are met with, broadly differentiated by their pathological conditions and their clinical features. The first of these is characterised by the existence of a definite and demonstrable lesion, consisting either of morbid new formation or of tissue degeneration. This not only leads to abnormal signs, as a direct result of the structural alterations, but to a series of symptoms due to perturbation of the functions of innervation. Such signs and symptoms bear a constant relation to the lesion which caused them, and may therefore be looked upon as secondary events, as they disappear when the primary change no longer exists. Disease under such conditions is called organic. To be distinguished from this is the second class of disorder under consideration. Here precisely the same symptoms may ensue, but they arise idiopathically, and are not the result of any pre-existing lesion or demonstrable tissue change, nor are they accompanied by any of the signs resulting therefrom. As in organic disease, the symptoms themselves are only modifications of the functions of the nervous system, but they are independent and possess certain peculiarities. They are induced by causes which do not give rise to structural change; they are characterised by extreme mobility and readiness to modification; the same influences which produced them may effect their sudden and complete recovery without a trace being left of their existence, and however long they persist, they do not necessarily lead to further derangement or danger to health and life. Such a train of conditions has been termed, a neurosis, or functional nerve-disorder.

Between these two forms it must be admitted that there is no fixed line of demarcation, and that the definition of each is purely an arbitrary one; at the same time it may be conceded that for practical purposes, both on pathological and clinical grounds, such a distinction may be permitted. In both, if the symptoms differ in character and behaviour, they are identical in kind, consisting, as they do, of a modification of the functions of the nervous system, of increase, diminution or perversion of the intelligence, motility, sensibility, or sympathetic processes. In organic diseases these are the secondary results of a definite lesion; while in functional disorder they arise independently, constituting, as it were, a simple instability of natural acts.

It has, however, been maintained that the well-marked and often permanent abnormalities that are seen in some neuroses must be represented by structural change. In one sense this cannot be denied. Every healthy act or function we may hypothetically assume to be accompanied by molecular movement in the nervous centres. This, it is obvious, can never be detected or anatomically displayed. Should such acts or functions become disorderly, as during violent emotion, we may conceive an atomic change to accompany them, but this we cannot hope to demonstrate. Finally, should this irregularity become persistent, it is called disease, the symptoms of which are apparent, but the molecular movements which led to them would for ever remain unknown. Such a theory may explain what is known as functional disease, which may be described as a persistent modification of the condition in health, without further structural alteration than that accompanying functional activity.

Whether prolonged functional disorder ever leads to permanent tissue-disorganisation is not known. Experience tells us this is not necessary, as persons may suffer for years from symptoms of the greatest severity which may rapidly and completely recover. On the other hand, the progress and termination of certain cases of chronic hysteria and other neuroses would suggest that it is not impossible, as their symptoms and termination seem only to be explained on such an hypothesis.

It does not of course follow that because an organic lesson is not demonstrated the disease in consequence is a functional one. There are many disorders in which the present state of science fails to indicate any objective changes, yet which are not placed under this denomination, as their symptoms and termination seem to indicate that although we cannot prove a structural degeneration, in all probability such exists. In this, as in all other difficult circumstances, in deciding to which category any particular malady is to be placed, we must be guided by the general features of the case, and by the application of broad principles.

It would therefore appear that there is room for a special class of disorder, not strictly defined, it is true, to which, for convenience, we give the name of neurosis. It is an affection of universal prevalence, and if not actually endangering health or life, is accompanied with the profoundest suffering and misery. Of the exact nature of the condition we know nothing, for, as already pointed out, not only are the anatomical alterations incapable of demonstration, but from their character it is likely that they will always remain so. The manifestations only are apparent, and although these often are severe and protracted, they happily offer encouraging hopes for medical treatment, as from their *nature* there is nothing to prevent complete recovery.

Although the pathology of the neuroses has not advanced, and still remains exposed to the attacks of hypothesis and speculation, our general information concerning the phenomena presented by this class of diseases has steadily progressed, and a review of the work before us to a certain extent indicates the additions to science which have been made during the last quarter of a century. Twenty years ago, when Axenfeld published the first edition of his book, as M. Huchard points out, an exact classification of the neuroses was impossible, our general knowledge of nervous diseases was confused, their study was the more difficult as most of the affections were ill defined, their symptomatology was vague, their diagnosis undecided, their etiology obscure, and their treatment too often left to chance or empiricism. Since then our acquaintance with these details has made great strides. In addition to the three classes of neuroses described by Axenfeld, namely those of intelligence, sensibility, and motility, we now recognise those of the vaso-motor system. On some questions our information has undergone little change and the description of tremor, paralysis agitans, the peripheral and visceral paralyses, neurasthenia, &c., remains the same as it did twenty years ago. Other subjects, however, have received considerable development, and among these may be mentioned the neuralgias in general, and in particular, migraine, angina pectoris, vertigo, functional spasms, contracture, epilepsy, chorea, and hysteria. In all of these, if no actual discoveries have been made, our knowledge has been extended, and their nature has become more fully understood. On the important subject of hysteria the Editor has more especially found it necessary to make large additions to the text of twenty years ago, and in the chapter devoted to this subject is embodied the discovery of the various phenomena connected with hystero-epilepsy, hypnotism, and metallotherapy, which of late years has attracted the attention of the profession, especially in France. In addition, our information on this neurosis has been augmented by researches on its visceral symptomatology; the relations between it and other morbid states; its vaso-motor and secretory disorders; its convulsive phenomena, contractures, and paralyses; its intellectual and mental abnormalities, and its treatment. In an affection so universal and always so distressing—although in England we do not see its worst forms—all such improvements in knowledge must be of the greatest practical importance. M. Huchard endeavours more especially to penetrate into the mental and intellectual peculiarities, and to analyse the conditions of the character and mind, in short, to study the patients as well as the disease. The Editor also gives treatment the fullest consideration, and he remarks that many distinguished men occupy themselves with scientific research on disease, but place therapeutics in a secondary position. The patient does not look upon the question in this light, and M. Huchard declares himself in this respect an ally of the sufferer. In this we entirely sympathise with him, and there can be little doubt that of all the ills to which mankind is doomed, the neuroses are those which cause the greatest amount of suffering, and at the same time hold out the most encouraging prospects to treatment. Although many such disorders become chronic and intractable, there are many in which a physical and moral *régime* is attended with the greatest success, and it is probable that as the affection is better understood and more easily recognised, prophylactic and curative measures will diminish a large amount of misery.

The diagnosis of the neuroses is frequently attended with great difficulty, as their origin is obscure, and the symptoms may simulate in every respect those of organic disease, or may be complicated with them. It is clear that the treatment suitable for the one is positively contra-indicated for the other. Thousands of persons have drifted into a state of chronic invalidism who, had their true condition been recognised, might readily have been cured; and, on the other hand, many have been neglected, or even ill-treated, on the supposition that they were suffering from a trifling or imaginary disorder, when in fact they were the victims of an intractable disease. Such a work as that before us is eminently calculated to dispel the clouds from the horizon of ignorance, entering as it does into the whole subject in a liberal spirit of scientific and practical enquiry.

M. Huchard, while giving credit to the observers of all nations for advancing the science and practice of neurology, claims for France a pre-eminence in that particular, recording in proof the names of a phalanx of distinguished men who will remain imperishable in the annals of medicine. In some respects this statement cannot be altogether denied. At the same time, when we glance over the history of our profession in past and present times, and review the really great discoveries which have influenced its progress or established our knowledge, it is questionable whether that country is entitled to more than an equal share of honour. In the investigation of the neuroses it may be admitted that the French have taken a specially leading position, and this may be explained by the fact that this class of disorder is more prevalent, more widely distributed, and presents more severe forms, than with many other nations. It would, however, ill befit those regions of the world which are more fortunate in the comparative absence of such nervous maladies, to grudge any credit to their confrères for the advantage they possess in a larger experience, which they have so eminently utilised. They therefore welcome a work like the present, which indicates the desire, common to all truly scientific, to advance knowledge, and extend practical usefulness.

A. Hughes Bennett.

The Applied Anatomy of the Nervous System.—By Ambrose L. Ranney, A.M., M.D., Adjunct-Professor of Anatomy, New York, &c. pp. 500. 8vo. H. K. Lewis, London, 1881.

The purpose of this work is indicated by the full title "The Applied Anatomy of the Nervous System, being a study of this portion of the human body from a standpoint of its general interest and practical utility, designed for use as a text-book and a work of reference." Works of this nature have appeared only within a comparatively recent period, and the necessity for them becomes gradually more manifest as anatomy and physiology become every day more and more of independent sciences. To the last generation of medical men, anatomy meant almost entirely surgical anatomy, and even yet there lingers something of that tradition. Such a work as the last edition of Quain's Anatomy is a magnificent protest against such a notion, doing full justice as it does to the anatomy of the nervous system and of the thoracic and abdominal viscera. But the old system, notwithstanding its narrowness, had its good side. What anatomy was retained had a practical bearing, and exerted, so far as it went, its due effect on the practice of medical men. It was all applied anatomy. Can we always say this at the present time? We fear that the rank and file of students, those namely who are to form the bulk of our practitioners, fail to see any practical bearing in the greater part of the anatomy and physiology of their first two years of medical study. As a consequence they do not digest and assimilate these subjects but simply swallow them, more or less completely as the case may be, with a view to successful disgorgement at their examinations. The process is fatal to anything like a systematically scientific diagnosis and treatment of disease. The evil has been recognized, and works more or less allied to the present have been published in the various departments of anatomy and physiology, their avowed purpose being to serve as links between the abstract sciences and their concrete embodiments in clinical medicine and surgery.

Detailed criticism of such a work as this, is of course impossible, containing as it does an immense number of facts, theories and explanations. Criticism is, however, much simplified by the fact that there is no claim and no attempt at originality or even at independent observation. The author has found an abundant material ready to his hand, and the question therefore resolved itself into one of clear and accurate statement with methodical and perspicuous arrangement. He has succeeded on the whole in producing a readable book, the diffuseness and iteration of which results no doubt from its having been originally delivered in the form of lectures before the medical students of New York University. Throughout the book the author makes very large use of Hilton's classical work on rest and pain. The principle of a common nerve-supply in cases of localized pain associated with distant disease, is one of immense importance in diagnosis and treatment; and its systematic and explicit recognition by Mr. Hilton constituted a distinct advance both in neurology and in practical medicine. At the same time it is well that the student who reads this work or Mr. Hilton's should remember that the nerve-fibres within a nerve have as little direct connection as a couple of telegraph wires that run on the same posts. We have got an important, a most important practical fact, but we are in reality no nearer the explanation of these strange cases. It must necessarily be a matter not of nerve-fibres but of nerve centres.

The first fifth of the book is devoted to the brain. It is somewhat wanting in cohesion and arrangement and is frequently by no means clear owing to a confusion of nomenclatures. For example the supra-marginal convolution of Turner is also called the supra-sylvian convolution and the parietal lobule, while the term supra-marginal convolution is also applied to the portion usually termed superior parietal lobule. Functions and centres are discussed first, then topography, and finally functions and centres again. To the important subject of the vascular supply of the brain there is only the most meagre reference.

The two hundred pages devoted to the cranial nerves form unquestionably the best part of the work. The account of the innervation and movements of the eye-ball and specially of the iris, along with their clinical associations, is clear and full, although in a work like this we are somewhat surprised to find no reference to Charcot's cerebral decussation of the optic nerve-fibres. Again the anatomical and clinical discussion of the facial nerve is most detailed, but we find no notice of the peculiar character of the facial paralysis usually occurring in hemiplegia. Considering the importance of the principle and the light which it throws on many cases, we should have expected a full discussion of Dr. Broadbent's principle of the bilateral representation of muscles acting bilaterally, or as we ought rather to express it with Dr. Hughlings-Jackson, the bilateral representation of bilateral movements. At pp. 245 and 265 it seems to us that the efferent inhibitory action of the vagus is confused with the afferent depressor action.

The last part of the work is devoted to the spinal cord and spinal nerves, and is upon the whole both full and accurate. We should have liked to see a careful analysis of the various reflexes used in testing the condition of the cord at different levels. English readers will do well to note that Dr. Ranney uses the term postero-lateral column to mean the posterior part of the lateral column, not as is usual the lateral or external part of the posterior column, sometimes called the root zone of Charcot.

The work is freely illustrated, principally by the beautiful plates of Sappey and of Hirschfeld. A few diagrammatic figures are added, some of which are really useful, but some are useless, and others, such as Fig. 65, are both useless and incorrect.

As already indicated we approve most cordially of the purpose of this work, and doubtless in a second edition Dr. Ranney will by condensation and addition much improve its present form.

JAMES ANDERSON, M.D.

Lehrbuch der Neurologie.—By Dr. G. SCHWALBE, Professor of Anatomy at the University of Königsberg. pp. 740. 8vo. Erlangen, 1881. Eduard Besold.

THIS work, which forms the second part of the second volume of Hoffmann's 'Human Anatomy,' has been for some time in the hands of neurologists, and perhaps the best criticism passed upon it is the fact that we are already familiar from our recent English text books with many of the original observations and wood-cuts that appear in it. The first forty pages are devoted to an introductory account of the nervous system generally, with a description of the minute anatomy of nervous tissue. Terse, clear and pointed, with all the advantage of a good typography, varied so as to express to the eye the importance of the different heads, this introduction is a fair specimen of the rest of the work. A short section on the methods of neurological research is specially valuable and interesting. These methods he divides into morphological, physiological, and pathological. Under the first head he discusses the purely anatomical method, showing the restrictions under which this method labours in the case of the nervous system, and the great assistance derived by it from comparative anatomy and from development. The physiological methods of section or extirpation and stimulation are next referred to, leading naturally to the experiments performed for us by nature in disease and the results of disease or injury, including the Wallerian degeneration and its interpretation.

Passing on to the spinal cord, we are at once struck by the fact that while the work is strictly anatomical, it still, as we should expect from the introduction, takes full cognisance of the results of physiological and pathological research. The result is, that this work, more than any other on the anatomy of the nervous system that we have seen, can be most cordially recommended as a suggestive guide to those engaged in the study of nervous diseases. A marked feature of the work is, that at the end of each section we have a copious bibliography well up to date, a point that will be, we doubt not, fully appreciated.

The account of the brain and its structure, with the deep origins of its cranial nerves, will be at once recognised as the most important part of the work. As is now customary, the developmental rather than the anatomical arrangement is followed. Any inconvenience arising from this is easily overcome by a short preliminary account of the anatomical subdivisions. This part is enriched even more than the rest by numerous original plates, showing vertical as well as horizontal sections of the brain. Considering the great practical importance and also the difficulties in interpretation of the physiological experiments of Fritsch, Hitzig, and Ferrier, Professor Schwalbe's chapters on the variations and the comparative anatomy of the cerebral convolutions, will be welcomed and read with interest. Specially important are the sentences with which he closes this section, " We are at the end of our comparison, without sure results for the comparison of the cerebral convolutions of primates with those of other forms. This, I believe, need cause no surprise when we remember that in the case of the two great groups of mammalia compared, namely, the Primates and the Carnivora and Ungulates, we have to do with divergent developmental forms. In the group of Primates we see development commence with almost smooth brains, which in their collective faculties nevertheless stand incomparably higher than the brains of carnivora and ungulates. In each of the groups then there appears an independent convolution of the cerebral cortex on an already markedly different ground-form. It is therefore impossible accurately to compare the Primate brain with other mammalian convoluted brains, and I believe that for this reason the attempt to find the fissure of Rolando in one or the other fissure of the carnivorous or ungulate brain will have but little successful outcome." It is in the doctrine of evolution and dissolution, as applied psychologically by Herbert Spencer, and physiologically by Dr. Hughlings-Jackson, that we possess a clue to the mazes of mental and cerebral development. But in applying the doctrine we have to bear well in mind that we are dealing not with a ladder, step above step, but with a tree from whose trunk branch after branch strikes off.

The most valuable and most original part of the work is that devoted to the medulla oblongata and the great cerebral ganglia, with their relations on the one hand to the spinal cord, and on the other to the cerebral cortex. The numerous and beautifully executed wood-cuts introduced to show the structure and relations of these parts, constitute a most definite and valuable addition to neurology. Here again the statement of physiological and pathological results alongside of, but in no way confused with, anatomical facts, will be found specially valuable. In passing we may refer to the exhaustive bibliography of literature on the brain, to be found at p. 768.

While the last two sections of the work, devoted to the cranial and spinal nerves in their course and distribution, show fewer marks of original work than the preceding, they contain a full and accurate account of these nerves, written in a clear and easily followed style, and well illustrated by the plates of Hirschfeld and Leveillé.

We cannot but think that a mistake has been made in rendering this work incomplete through the omission from it of the vascular system of the brain and spinal cord. For this we are referred elsewhere, and perhaps it was unavoidable in view of the fact that the work before us forms only part of a large systematic treatise on Anatomy. Something is done to fill the gap by a few pages giving the blood supply of the various divisions of the nervous system, and also in the description of the meninges. The work would be very greatly improved by the addition of an index, which would add much to its utility as a work of reference.

JAMES ANDERSON, M.D.

The Field of Disease, a Book of Preventive Medicine.—By B. W. RICHARDSON, M.D., LL.D., F.R.S. 1 Vol. 8vo. London, 1883. Macmillan & Co.

UNDER this title Dr. Richardson has addressed a bulky volume of 1014 pages to "the intelligent reading public," in the belief that they "wish to know the leading facts about the diseases of the human family." In accomplishing his task the author has been careful, as he tells us, to "avoid the new names and terms which have recently stolen their way into the literature of medicine, preferring the old historical terms as not only familiar, but far more correct than the new ones." It is a bold resolve, and whatever may be thought of its wisdom in general, is certainly unfortunate in the present instance, inasmuch as it invites attention to some of the chief blemishes of Dr. Richardson's book. The old pathologists, in spite of their metaphysical notions and fantastical nomenclature, were graphic and accurate describers of clinical symptoms. "The Field of Disease," on the contrary, is neither graphic nor accurate in this particular. To speak only of the department of nervous disease, which is neither better nor worse than the rest, it is surely but a lame description of apoplexy to say that "it means a blow or sudden shock by which a person is stricken down," adding no further account of its paralytic symptoms than the statement that it is "followed by some deficiency of power or partial paralysis, which may be permanent, but from which there is often a fair recovery." Again, it is somewhat vague to speak of sclerosis as a "condensation of the brain substance, extremely similar to what has been before us when treating of the disease of the lungs, known as fibroid disease." What is added regarding its symptoms, does not contribute to any more precise notion of its natural history or modes of incidence. "It may come on in early life, it may come on in later life, and it may be one of the degenerations of old age. When it is present in children they lapse into imbecility. When it attacks the aged it leads to failure of the vital powers and to the second childishness and mere oblivion."

But it is not always that the book is thus uncertain and featureless. Witness, for example, the account of anæmia of the brain as differentiated from congestion. The question involves so nice a point of diagnosis, that the passage may be worth quoting. "Congestion of the brain," it appears, is "due to excitement and over mental work, as also to alcohol, passion, worry, and excessive study. The opposite condition, known as anæmia, is produced by mental and physical exhaustion, by insufficient food, by long continued physical work and fatigue." Striking out, therefore, the factors common to both—for over-mental work and excessive study may be taken as the equivalent of mental exhaustion and fatigue—we have excitement, over-mental work and worry combined with alcohol to produce congestion, while physical exhaustion together with insufficient food produces anæmia. We are further told that while anæmia produces ringing sounds in the ears and somnolency, congestion, "in its milder and more common forms," (forms, that is to say, of which, by the hypothesis, morbid anatomy knows nothing), "gives rise to a sense of fulness in the head, dizziness, restlessness, and desire for change, with sleeplessness and irritability of mind," a condition which "often repeated may lead to diabetes or paralysis." It may sound very probable, but is not all this the pathology of the fireside, rather than of the post-mortem room?

Much more might be quoted to like effect, the description of tubercle, of meningitis, of cerebral softening, of functional nervous disease. Each successive page is a new surprise. As we read of chorea, that it is a disease "whose phenomena present themselves at all ages," (with no mention of children or of females as its peculiar subjects), and are informed that "recovery sometimes occurs;" or encounter the striking statement, that the pleura is "that membrane which can so easily be stripped from the surface of the lungs," it is difficult to withhold a suspicion that the author himself is hardly as familiar as he should be with some of the matters he undertakes to simplify. And yet we are assured, more than once or twice, that it is from Nature herself that Dr. Richardson is drawing his descriptions. He has not only discovered a new form of phthisis "peculiar to those who indulge in alcohol," and which (without describing it anatomically) he proposes to call "alcoholic," but chance once threw in his way a quite unique case, which he uses as his warrant for the rather bold statement, that "extreme congestion of the pia mater is a cause of delirium tremens." It is so characteristic of the author, and of the sketchy, anecdotal style of his book, as to be worth quoting. A man committed suicide by casting himself under a railway carriage. "Within four minutes after death," Dr. Richardson saw that man's pia mater "lying like a velvety pile over the brain, and within the convolutions, while still the fumes of whisky were most readily detectable from it." Nothing is said of the grounds of the diagnosis, the nature of the injury which thus laid bare the pia mater, or of the post-mortem examination which so speedily followed. Only one bold touch is added. "In such examples the membrane, after many excitements, becomes firmly attached in parts to the substance of the brain."

It may be truly urged, that to give a faithful description of the facts of medicine in popular phrase is a task of no small difficulty. But here one is reminded of the dramatic author, who when censured for the badness of his composition, and pleading in excuse that it was not easy to write a five act tragedy, was met with the reply that it was quite easy *not* to write a five act tragedy. And it is quite easy not to write a popular account of practical medicine, especially easy, it might be supposed, for Dr. Richardson, busy with other subjects where his authority is undisputed. The general reader is already surfeited with popular accounts, and weary of being studiously talked down to. For any information he may require on medical subjects he will do wisely to consult the very same sources of knowledge as attract professional students—the works of recognised teachers like Sir Thomas Watson, or even some such text-book as the admirable and lucid compendium of Dr. Frederick Roberts.

And, indeed, in the later part of his book, and when no longer concerned with the precise anatomical connection between lung and pleura or puzzling himself about the distinction between meningitis and acute hydrocephalus (p. 218), the composition is so clear and masterly that it is difficult to believe that the authorship is the same. The part concerning "the cures and preventions of disease," contains much valuable information conveyed in simple and forcible language, which the best popular teachers may well envy. We have now no longer to criticise, but to listen; and read with amazement and concern of the anæmia, nervous depression, dyspepsia together with various derangements, circulatory and respiratory, which flow from the use of tea, coffee, tobacco and snuff. Not only, as it appears, is the cup that cheers, equally with the one that inebriates, laden with mischief for us, but the common emotions and passions produce distinct forms of disease. Intermittence of the heart, it seems, is *invariably* traceable to an emotional cause (p. 655), and even blushing (*erythema fugax*) is not so harmless as it looks.

In telling us that cheerfulness and contentment must be kept up, Dr. Richardson is but repeating what was said long ago by Mr. Squeers, but the wonder is to find the subject so closely studied that in "The Field of Disease" each particular passion has assigned to it its own peculiar danger, while articles of common consumption—and especially, be it noted, afternoon tea—are found to affect the human organism in ways but little suspected, producing, amongst other evils, nervous depression and low spirits.

Finally, and still with a master's hand, Dr. Richardson discusses the general principles of living in order that health may be preserved and old age postponed. We are not to grieve, not to hate, and not to be jealous. Yet, after all it is but a dreary picture, this passionless man, living in an evil and adulterating generation, mistrustful of the promptings of his special senses, for ever anxious and thoughtful about his own body and so encompassed by hidden dangers and subtle poisons that, with the very best intentions, Health can only be preserved by a diligent study of the voluminous literature which now goes by that name. Nay, it cannot be preserved anyhow in its hypothetical perfection; for the very concern and anxiety which this watchful regulation of self demands, is of itself a detriment and must in a measure defeat its own object. The smoker of tobacco and the afternoon tea-drinker may be sad spectacles enough, but sadder still, I think, is the contemplation of the man who refrains from emotion or sympathy on the score of his digestion, and with no better end in view than the prolongation for a little of his own lean and self-regarding existence, is continually worrying himself and his friends about the things of his body, the purity of his food and his drink, or the dictates of prudence in regard to a second cup of tea. The life is more than meat and the body than raiment.

OCTAVIUS STURGES.

rate of progress varies materially in different cases, being some times slow, at other times very rapid. From the observations already made, it seems to the authors probable that in some cases the neuritis may extend from periphery to centre, or attack by centripetal migration the spinal ganglia, and even the spinal cord itself. They recognise five different phases or types of degeneration, for an account of which it is needful to refer to the paper itself, and to the beautiful coloured drawings which accompany it. The types are thus described:—I. Fragmentation de la myéline en blocs. II. Fragmentation de la myéline en boules. III. Fragmentation de la myéline en fines granulations avec atrophie discontinue de la fibre nerveuse. Etat variqueux, moniliforme. Renflements fusiformes. IV. Atrophie des tubes avec granulations ambrées dans l'intérieur des gaînes. V. Atrophie complète des tubes nerveux; vacuité des gaînes: headings which very aptly describe the characters of the degeneration. The authors then give a full account of four cases, in three of which there was acute bed-sore—the "decubitus acutus" described by Charcot—after hemiplegia, and in which the peripheral neuritis was well marked. It is a striking fact that in the first of their cases, and to a less extent in the others, although the patient only survived the apoplectic attack six days, the nerves in the neighbourhood of the bed-sore were profoundly altered, even to almost complete atrophy of their fibres. Wallerian degeneration never runs so rapid a course as this. Their last case is one of zona in the sixth and eleventh right intercostal spaces. The periphery of the eleventh nerve was found diseased throughout its entirety, while of the sixth nerve the fibres of the root and ganglion as well as the periphery were extensively degenerate. These observations are continued in later numbers of the same journal (May, p. 290, Sept. p. 180), the authors recording the clinical history of several cases, together with their microscopical examinations. Discussing the ætiology of Zona, they are of opinion that too much stress has been laid upon the root and ganglion changes and that microscopical examination has not been sufficiently directed to the nerve periphery, alteration in which was the one common pathological change in their own case of diseased 6th and 11th nerves. In all probability they think that the degeneration of the ganglion and of the posterior root, found in the 6th nerve but not in the 11th, was secondary to the peripheral inflammation, and that this latter it was which caused the herpetic eruption. Microscopical examination thus supports the view, which has been frequently expressed, that Zona may be due to a peripheral neuritis rather than to any central change. Cases VI. and VII. are most instructive examples of trophic and joint changes occurring in the course of tabes dorsalis—locomotor ataxy—and the authors call prominent attention to the fact that these changes were in each instance found to be associated with morbid states of the peripheral nerves supplying the affected parts. It had previously fallen to their lot, they relate, to make a careful examination of the spinal cord in cases of this kind, without being able to discover any pathological changes whatever in the anterior cornua, where by some it has been thought that the morbid condition may lie on which such trophic changes depend. They discuss at length the various reasons for attributing the arthropathies, and other trophic disturbances, of tabes dorsalis to central disease, and from a consideration of their own observations they conclude that it is at least highly probable that alterations in the peripheral nerves supply the real cause. At any rate it is a remarkable fact that in Case VII., where numerous lesions of nutrition arose in the course of tabes, to wit perforating ulcer, dystrophy of toe-nails, and arthropathic changes in a metacarpophalangeal joint, the peripheral nerves supplying the parts affected were the seat of extensive degeneration. That their observations are as yet too few to establish any precise dictum on this point the authors freely admit, and their conclusions are both cautious and provisional. All peripheral neurites do not of necessity give rise to trophic changes, but yet it is very obvious that trophic changes are in all probability provoked or caused by some primary alteration in the nerves. In Case VIII. there were developed numerous bed-sores, consecutive on myelitis from compression, which rapidly closed the scene in an old-standing case of angular curvature of the spine. The periphery of the nerves going to each one of the many sores was found atrophied and almost completely destroyed. But the point which above all they look upon as interesting in the examination both of this case and of others is the observation that the degeneration, extreme at the periphery, lessens gradually in degree as it is followed up the nerve-trunks, and that there is no evidence whatever of a continuous change extending in the reverse direction. And in those instances where the degeneration spread high up, the posterior roots were manifestly changed, although nothing abnormal whatever could be found in the anterior. The authors refrain from offering any explanation of this difference in the anterior and the posterior roots; they are content to record the fact which they have observed.

Thus, in their two somewhat analogous Cases, VIII. and IX. of myelitis dependent or spinal caries, with various trophic lesions in the last days of life, there were found well-marked changes in the posterior roots but none in the anterior roots of those nerves whose peripheries were diseased. How a lesion of the cord can exercise an influence on the periphery of nerves, and yet spare the trunks of these nerves, it is not easy to explain. One might assume that the degeneration spread continuously from centre to periphery, but the facts do not warrant this conclusion; and the authors are disposed to think that central lesion somehow predisposes to, but is not an immediate cause of, the peripheral neuritis which may arise in the course of disease of the central nervous system. The central lesion, on this hypothesis, so modifies the peripheral nutrition, that inflammatory or degenerative changes in the nerves are liable to arise from some accidental cause, such as undue pressure or other trifling form of injury. Be that however as it may, there is a radical difference between the centripetal march of a peripheral neuritis arising in the course of cerebral or spinal cord disease, and those centrifugal, or true Wallerian degenerations, set up by transverse section of nerve-trunks. That non-traumatic peripheral neuritis is a by no means uncommon affection the authors feel assured, since in a very short space of time, and with no great abundance of material, they have found no fewer than nine examples. They point out that the naked-eye appearances of the nerves are wholly insufficient to tell us whether a nerve is diseased or not, for these are usually normal, even when the microscope may reveal extensive pathological changes. And these changes essentially affect the ultimate nerve elements themselves, and only secondarily and at a much later date do they implicate the connective tissue. Following a variable course, unlike that described by Waller, the degeneration may amount to almost complete atrophy in the course of a few hours or days; and while no continuous connection can be traced between central nerve-lesion and peripheral disease, the neuritis beginning at the periphery seems to follow a truly centripetal march. Such are the main conclusions in the important contribution which deserves the careful study of all who are interested in the diseases, and the ætiology of the diseases, of the nervous system.

Déjérine on the Peripheral Nerve Lesions of Ataxia and on Peripheral Nervo-Tabes. (*Comptes Rendus*, 1883.)—This is a further and most important contribution, by an observer whose researches in the same direction are already widely known, towards a fuller appreciation of the changes met with in the peripheral nerves in the course of tabes dorsalis. Struck by the fact that although defects in sensation had been in several cases very various, the cord lesion was found in each to be the same, the author was led to make more careful examination of the cutaneous nerves, and the paper before us is devoted to an extended record of these two cases with complete clinical history and post-mortem examination. Both were well-marked instances of the disease, with ataxy, abolition of tendon reflexes, crises, and lightning pains, and in addition thereto were even definite areas of anæsthesia and analgesia of the trunk and limbs. The usual central changes were found, and microscopical examination of the cutaneous nerves led further to the discovery of grave alterations therein in the regions of impaired sensation. Thus in the first case we read that the nerve trunks presented exactly those appearances which are ordinarily seen some months after section, very few indeed of the nerve fibres having the normal physiological qualities. In the second case, that of a woman aged 55 who had pronounced anæsthesia of the legs, the cutaneous nerves showed extensive degeneration indicative of an essential parenchymatous neuritis. The anterior roots were perfectly healthy, but the posterior roots between their ganglia and the cord were markedly altered. Below the ganglia, however, that is, between them and the point of coalescence of the anterior and posterior elements of the mixed nerve, no such changes were to be found. And the ganglia themselves were also healthy, an observation on which M. Déjérine lays stress as an indication that the peripheral changes of the nerves were not the result of any morbid condition of their trophic centres. The author enters into a full discussion upon the nature of these peripheral lesions, combating the view that they are in any way allied to the changes seen in the optic nerves, and coming rather to the conclusion that they are the result of a true peripheral neuritis, starting and developing like the medullary lesions quite independently, and showing no connection therewith, either as cause or effect. Peripheral lesions are daily becoming more known, and although experimentally speaking true Wallerian degeneration has an existence beyond all dispute, it is none the less true that the cutaneous nerves may show serious degeneration without any central change, the known examples thereof becoming rapidly more numerous as observation is more frequently directed to their examination. In the present state of our knowledge it is impossible to speak with any degree of certainty as to the mode of origin of peripheral neuritis, or as to the precise influence it has in giving rise to some of the symptoms of tabes dorsalis, the retardation of, for instance, sensory impressions, the incoordination, &c., but there can be little doubt that it does play an important part in the disturbances of sensation which are so common, though so variable, in the disease. And confirming and strengthening these conclusions are the facts recorded in a later and most interesting communication from M. Déjérine on two cases of "peripheral nervo-tabes," in which there were many of the symptoms of locomotor ataxy ('La France Médicale,' 30th of October, 1883, No. 31), pains, incoordination, abolition of knee-jerk, anæsthesia, analgesia, and retardation of perceptions, where post-mortem no central lesions whatever could be found, the cutaneous nerves alone showing disease, having in fact undergone extreme degenerative changes, the result of peripheral neuritis. These cases are now only briefly recorded, for M. Déjérine intends to bring them forward in greater detail in a work on which he is at present engaged, and which will be looked forward to with much interest. He has found nothing like them in medical literature, and he places them on record with the object of showing how in some cases locomotor ataxy may exist independently of lesion in the spinal cord, and of suggesting for them the name, "Nervo-tabes périphérique," in contradistinction to that of "Tabes médullaire."

Johnson on Nerve-suture and Nerve-transplantation. (*Nordiskt Medicinskt Arkiv*, Band xiv. No. 27, p. 1.)—This is a contribution to the study of nerve-suture, both from clinical and experimental observation. The fifty-two cases collected by the author from various sources, and arranged in an admirable table, speak very forcibly of the benefit usually derived from the suture of divided nerves. M. Johnson has himself conducted a large number of experiments in the laboratory of the school of medicine at Stockholm, both on nerve-suture and nerve-transplantation, his observation being chiefly directed to a knowledge of the time when conductivity was re-established after the operation. This varied in different animals, being found on the 40th day in rabbits, on the 31st in dogs, and the 25th in fowls. It was not, however, until the 60th day that conductivity was restored after simple division of the sciatic in rabbits, and when no suture was applied. This was tested in each instance by a weak faradic current, or by some mechanical irritation, applied above the cicatrix. Microscopical examination, moreover, showed a like difference in point of time, nerve fibres being in rabbits clearly traceable through the cicatrix on the 40th day after suture, on the 60th day only after simple section. The fibres in the latter case seemed rather better developed than in the former. Regeneration took place with greater speed in dogs and fowls. M. Johnson points out that nerve-suture is free from danger, neither trismus nor tetanus having followed it in any case; and he strongly recommends the practice, there being no question that restoration of function is thereby accelerated. It is less painful, and less likely to set up trophic disturbance, if the suture is indirect than if it is passed through the nerve-trunk itself. In three cases, where pieces of the sciatic had been resected in fowls, he endeavoured to transplant portions of nerve, taken in two instances from the sciatics of other fowls, and in one instance from the sciatic of a rabbit. The animals were killed on the 28th, 24th and 23rd days respectively, but although the grafts were in each case firmly united, conductivity was not the least established, and, as might indeed have been expected, microscopy revealed almost complete degeneration of nerve-structure in the engrafted fragments, in marked contrast to the condition of the nerve itself. In addition to the large number of collected cases, the paper is enriched with an account of other experimental observations, and with a full bibliography on the whole subject of nerve-injury and nerve-suture. HERBERT W. PAGE.

Two Cases of so-called Spastic Spinal Paralysis. In a reprint from *Il Movimento medico-chirurgico*, Professor Bianchi details two interesting cases, and gives an account of the changes found in the nervous system after death. The first case was that of Giuseppe Ippolito, aged 67, who in his occupation as a baker had been exposed to sudden changes of temperature. Had been a soldier, and been punished altogether to the amount of 830 blows. At 25 years of age had syphilis. In 1852 felt a weakness of the lower limbs with rigidity, worse on rising in the morning, or on getting up from his chair. Had to use first a stick and then a crutch to get about. No other ailment, except slight formication. By-and-by tremblings in the limbs on walking so that he required two crutches; and loss of power over the joints, with extreme rigidity. When he came under the notice of Professor Bianchi, he suffered from no loss of sensation nor any muscular atrophy, except a certain loss of rotundity about the limbs. The man lay in bed unable to move, the thighs flexed on the abdomen, the legs on the thighs, the feet in slight dorsal flexion and abducted, the knees so tightly compressed against one another that he had to put a pillow between to prevent the suffering. The cutaneous reflexes natural, or rather increased. Electric examination showed no changes of importance. Before death changes occurred in the sensibility; and there was paralysis of bladder and rectum.

On necropsy, the spinal marrow was softened and reduced almost to a pulp from the lumbar region as high as the third dorsal nerves. On section the bundles of grey matter normal, except that the columns of Goll shewed a noticeable change of colour observed after the use of both hæmatoxylin and picro-carmine staining. On account of the great length of time during which the disease persisted (31 years) no certain conclusions are drawn by Dr. Bianchi from this case.

Case 2 is more important. It is that of a boy, G. Finorelli, aged 12, a deaf mute of deficient intellect. At 9 years of age began to be unsteady in his gait, his steps being constrained and the body inclined to right or left, in order to enable him to lift his feet, the points of which he dragged along the pavement. When he rested on the ground, the lower limbs trembled, and when he got into bed, they became rigid. The disease made rapid progress, and soon the boy could not walk at all, though he used his hands perfectly. No rigidity of the superior extremities, but lower limbs rigidly contracted. Soon, however, difficulty arose in using the left upper limb which was followed by complete immobility. Two or three months later, rigidity of right upper extremity. At this time Dr. Bianchi had the opportunity of watching the patient. Spasms of the trunk and contractions of the muscles of the neck followed. The lad seemed to have pain, for he cried out when strong contractions of the lower limbs occurred. The lad now lay always supine, the lower limbs immovable and rigid as bars, the muscles hard and raised. The rigidity which was greater on the left side, increased, the leg being forcibly extended on the thigh, the thigh on the pelvis, and the foot in the equino-varus position crossed over that of the other side. The first phalanges of the toes extended on the metatarsus, the second and third strongly flexed. On attempting to make movements, they could not be executed, and rigidity was increased, the stiffest muscles being the quadriceps extensor, the gastrocnemius, the posterior tibial, and the flexors of the toes. No tendon-reflex when the limbs were rigid, but when a little relaxed the patellar-reflex was increased, and sometimes knee clonus came on. The cutaneous reflexes became diminished. The cremasteric reflex nearly disappeared. At the same time tickling of the sole increased the contraction, the lad cried at the same time flexion occurred. When the coverlet was raised, the contraction increased, till it became painful, and drew cries from the lad. It was very difficult to judge how much the tactile sensibility was affected, the boy being dumb and unable to move. The left upper arm was forcibly laid along the side of the thorax, the forearm pronated and firmly flexed on the arm, the fist extended, the fingers flexed, the thumb folded in the fist, and the proximal phalanges extended on the metacarpals. The muscles well nourished, the biceps hard. The right upper limb could be somewhat moved, but efforts to do so required much more force than natural.

On electric examination, the muscles could be made to contract in the lower limbs by the induced current, but the contractions so induced did not overcome the antagonistic contractions of the muscles in spasm. The induced current also caused the muscles to contract in the upper limbs, but on the left side every application of the current on the forearm increased the rigidity of all the muscles contracted in mass, and from the position of the limb it was not found possible to act on any of these.

By the galvanic current results were got from 12 to 26 elements, the fewest elements being required on the right side. Eight to ten days before death, the contraction of the muscles of the neck increased, piercing shrieks were emitted, passed along as it were from tract to tract; the temperature rose, the face became glassy and expressionless, some attempts at vomiting ensued, slight strabismus with somnolence, coma and death.

On necropsy, two facts bearing on the deficiency of intelligence were observed. The first was the incomplete development of the temporo-sphenoidal lobe, especially of the first temporal convolution. The second was the prolongation of the parieto-occipital sulcus on the outer surface of the hemispheres, through the defect of development of the first fold of the passage of Gratiolet, very much as is observed in the brain of certain monkeys. The occipital lobes were little developed, but there was an exaggeration in the development of the parietal lobes, so that the brain appeared short and high, and the fissure of Rolando less oblique.

The spinal cord cried somewhat on section by the knife. When ections were hardened for three weeks in solution of bichromate of potash and coloured with picro-carmine, two sets of alternations were observed. One of these was precisely and systematically limited to the pyramidal bundle of fibres of the two sides: the other, without relation or apparent continuity with the former, was limited to the central canal and its surroundings. In drawings from a series of sections of the cord through the medulla oblongata at the middle of the olivary body, at the bottom of the olivary body, between the 1st and 2nd pairs of cervical nerves, at the level of the 6th cervical nerves, in the dorsal and the lumbar regions, the degeneration of the fibres making up the pyramidal bundle was consecutively traced, as also the relations of the same at different levels. From these it is seen, that the degenerated spot which was pretty large in the lumbar region was reduced considerably in the cervical, by the crossing over of some of the pyramidal fibres to the outer side of the fissure. The direct cerebellar bundle of fibres was also larger there. In the medulla oblongata no lesion of any moment was found, except in the pyramidal fibres. Figure 6 is very instructive, showing, as it does, in the section of the right side of the medulla oblongata at the middle of the olivary body, that the only abnormal portion is the anterior pyramid.

A section of the brain shewed a sclerosed nucleus of a grey yellowish colour, much harder than the surrounding substance, of the size of a bean, in the very centre of the centrum ovale, of which only a very small zone remained normal and divided the sclerosed nucleus from the cortical grey substance. This nucleus was prolonged backwards, and was found to extend to the fibres of the 2nd and 3rd frontal convolutions. Still further back it occupied the expansion of the *corona radiata* of Reil, corresponding with the ascending frontal and parietal convolutions, and was prolonged into the internal capsule which was degenerated in the posterior half of the anterior segment, the *genu* and a large part of the anterior portion of the posterior segment. A similar lesion was found on the other side, the degeneration being confined to the motor zone, and the nuclei of the base and the grey substance being entirely spared. In short the motor portions were sclerosed and the sensory portions were normal. Histologically, the degenerative changes consisted of an enormous increase of the connective tissue, developed entirely at the expense of the nerve-tubes; of an increase of the nuclei and of the lymphatic corpuscles; of a diminution of the nerve-tubes, many of which were, however, well preserved, some having the axis-cylinder most minute, and others the nerve-sheath sclerosed with a great diminution of the medullary sheath and the axis-cylinder much increased, these last however being rare.

In the internal capsule, however, a different appearance was observed. Where the degeneration was most advanced, there was observed the same fibrous connective tissue found in the pyramids, with scarcely any nervous fibres for long tracts.

In the intermediate zone there were seen in the midst of a granular quasi-amorphous tissue an immense number of lympathic corpuscles and nuclei, with a development of capillaries, and no nerve-fibres. After further description of the changes found, Professor Bianchi makes some remarks on Schulz's question: Is there a primary sclerosis of the lateral columns of the spinal cord? and refers to a case published by Zucha to show that the symptoms may all be present without a lesion of the pyramidal bundles, the disease being confined to the brain. For this reason Dr. Bianchi suggests that the disease should be called *spastic paralysis*, and not *spastic spinal paralysis.* On this we would remark, that if it were called *sclerosis* of the *pyramidal tracts*, or sclerosis of whatever portion of the brain may be affected, or both, we should have a really scientific name of the disease. *Paralysis* is no more the name of the disease than *dropsy* would be of mitral incompetence, though each is a symptom in its own place. If there were any softening, that ought to be named also.

La Psichiatria, la Neuropatologia e le scienze affine is the title of a new quarterly journal devoted to inquiries in the domain of the nervous system. It is under the directorship of Professor Buonomo, medical director of the asylum at Naples, and is edited by Dr. Bianchi, assisted by Drs. Andriani, Armanni, Cantarano, Fede, and Nicolucci. This journal will take the place for the Southern Italian Provinces filled by the *Archivo delle Malattie nervose* of Milan, and the *Rivista sperimentale di Freniatria* and other journals published in North Italy.

Professor Buonomo leads off in the first number with an article on the value and importance of a study of nervous diseases, pointing out that the general acceptance of the theory of evolution has revolutionised this (as indeed it may be said to have done all other) department of inquiry, so that human psychology can no longer be the result of the self-introspection of the philosopher, but must be founded on comparative anatomy and comparative physiology. Professor Buonomo goes on to say that insanity is the special study of the medical psychologist, and to remark on the vast importance of its sphere in determining questions connected with mental capacity and responsibility, more especially in connection with penal legislation and civil legal relations generally.

Professor BIANCHI has an interesting and important article on *Functional Compensations of the Cerebral Cortex.* The various theories of accounting for these are considered shortly, and Dr. Bianchi goes on to formulate certain conclusions, detailing the experiments on which he rests them. The subject is continued in the second number of *La Psichiatria*, some 40 pages of which are devoted to it. The monograph is well worth reading, as was to have been expected from so enthusiastic an enquirer, but our space allows us only to state his main conclusions. The first proposition is the following: Recovery from the paralysis consequent on an extensive destruction of the motor zone in dogs is only apparent and partial, only locomotion and acts correlative therewith being restored; every other movement in different conditions remains permanently abolished (for months) and power is permanently diminished.

2nd. The parts surrounding the excitable points of the motor zone of limbs (which portions have remained perfect after a limited destruction) may to a certain extent compensate the functional power of the part destroyed, though without acquiring electric excitability when acting on the parts whose centre has been removed.

3rd. Locomotion is not determined by special centres in the brain of dogs; in these animals the cortical motor zone may be destroyed without any lasting disturbance in locomotion.

4th. In dogs, when the motor zone of one side has been widely destroyed, the opposite hemisphere, and especially its motor zone, exerts a controlling power on the limbs of both sides.

5th. If, during the period of development, the cerebellum has been in great part destroyed, the cortical motor zone may in dogs perform its functions.

Under the consideration of this proposition certain questions arise regarding the sense-organs, and Dr. Bianchi states the following conclusions:

(*a*) The fibres of the retina of each eye go in large part to the cortical centre of the opposite hemisphere, but to a smaller extent to that of the hemisphere of the same side.

(*b*) The cortical visual centre of dogs is very extensive, comprising all the second external convolution from its anterior extremity as far as the occipital lobe close to the latter, and part of the first and third external convolution.

(*c*) Compensation is made in the portion remaining entire when the cortical centre of the same side has been partially destroyed. It is very doubtful whether the centre of the opposite side contributes thereto.

In order to establish these conclusions some sixteen experiments are detailed, and under the head of general considerations, the two following propositions are laid down.

(A) A cortical paralysis is the graver, the more extensive is the lesion of the anterior lobes of the cerebrum, within certain limits not yet determined.

(B) The central elements of innervation for a given organ of motion are diffused over all the motor zone, and are merely found more condensed in certain points, which constitute therefore the so-called excitable zones, which up to the present time have been erroneously considered as the only cortical centres corresponding with special groups of muscles or with a given limb.

Thirteen corollaries which Professor Bianchi believes himself justified in drawing from these premisses, bring to an end this valuable paper.

Professor NICOLUCCI contributes an article of some merit on *the brain of man, considered under an anthropological aspect.* After shewing that according to the recorded nomenclature, there are 44 convolutions in the brain, and that 16 of these are in the frontal lobes, he says this plainly shows the great predominance, exerted by the frontal lobes over the other lobes of the human brain, and consequently the highly important part they play in the exercise of the encephalic functions. But Professor Nicolucci's inquiries have led him to results far less certain than those commonly believed, and he says: "I do not hesitate to declare that," the conclusions generally accepted regarding the localisation of the cerebral functions "are yet very doubtful, and even to some extent erroneous. It is also to be remembered that the experimental investigations of this complex question have been only commenced, and it is reserved for future observations to assist in forming conclusions less uncertain and more trustworthy." The writer then goes on to show that observations are very deficient, up to the present time, regarding the weight and quality and convolution-markings of several human races, as for instance, Negroes, Arabians, Algerine Berbers, Chinese, &c. So far, however, as the facts are known, very great differences are found in the weight, and even in the disposition, of the substance of different human races; and these, in Professor Nicolucci's opinion, have not received the attention they deserve.

The remainder of the first two numbers of *La Psichiatria* is made up of numerous reviews. A. RABAGLIATI.

Archives of Psychological Medicine and Neurology. Kharkof, 1883. Parts 1 and 2.—It is significant of the lively interest excited by recent advances in the domain of neurology that a new quarterly journal, devoted to this subject, should make its appearance in a provincial town of Southern Russia. Great credit is due to the energy and initiative of its editor, Dr. P. Kovalevsky, who has succeeded in filling his two first numbers with much interesting material. As the journal is printed in the Russian language, its circulation will, of course, be limited to the Russian Empire. Any matter of striking originality or importance that it may contain is sure, however, to find its way to us through Germany. A brief summary of a few of the principal contributions to the two parts already issued, will serve to give the readers of BRAIN some idea of the scope of the undertaking.

The editor describes two cases of old-standing destruction of the eyeballs associated with wasting of the occipital lobes. In one, the right eye was alone affected, having been destroyed by an injury some twenty years before the patient's death. There was conspicuous asymmetry of the back part of the hemispheres, owing to atrophy of the convolutions of the right occipital lobe. In the second case, both eyes had been withered for more than twenty years, and the wasting of the brain was bilateral.—Dr. Orshansky furnishes the results of an experimental inquiry into the effects of anæmia in modifying the excitability of the cerebral cortex.—A most interesting account is given of the public lunatic asylum of Bessarabia by the medical officer in charge of it. The state of things described is quite equal to that which formerly existed in some of our work-houses. The one hopeful feature is that it should be possible, in a country like Russia, to publish so frank and outspoken a report. Improvement cannot be far off when once the light of day is admitted into such dark places.—A clinical lecture by Dr. Pasternatzki of Warsaw deals with a remarkable example of major hysteria in an adult male, a cornet of cavalry, twenty four years of age. This patient presented the symptoms of complete hemianæsthesia, induced hypnotism and catalepsy, violent hystero-epileptic paroxysms arrested at will by pressure on a hysterogenic area, and all the train of extraordinary phenomena with which the writings of Charcot have made us familiar. He seems to have been cured by the application of cold water douches to the spinal column.—A considerable part of each number is taken up with abstracts of papers from foreign journals and reports of cases of medico-legal interest occurring in the Russian courts.

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In conclusion, it is only fair to add that the importance and usefulness of this new periodical are not to be measured solely by what it may add to our scientific knowledge. Those who are in some measure familiar with the conditions under which it is produced, and the public to which it appeals, will recognise and welcome it as a new window opened towards the West. Every such window comes to admit more air and more light than those who throw it open can foresee. E. Buchanan Baxter, M.D.

Archives de Neurologie.—In the November (1882) number of the 'Archives' Dr. Gellé commenced a series of articles which deal with the clinical history of Ménière's disease, and the diagnostic features of different forms of labyrinthine vertigo. He briefly describes the usual form of attack, and alludes to the clinical studies of the disease instituted by Vowry, Bonnenfant, Lhuissier, Léo, Féré, and Demars.

Ménière, Saissy, Moos, Politzer and Voltolini have described affections of the labyrinth, and especially lesions of the semicircular canals in cases of pronounced vertigo with deafness, chiefly in cases of cerebro-spinal meningitis. In Gellé's cases, apart from the auditory troubles and disordered equilibration, the patients enjoyed perfect health. In Ménière's disease the troubles of equilibration, the buzzing sounds in the ear, and the deafness cannot all be referred to lesions of the semicircular canals; if the patient becomes quite deaf, it probably indicates something beyond disease of these canals, and this becomes a certainty if the deafness remain after the disappearance of the vertigo. Deafness is a most frequent accompaniment and termination of Ménière's vertigo, and therefore in seeking for the pathogenesis of the chief features of this disease, the deafness and buzzing, we must look for lesions outside the labyrinth.

The physiological experiments of Paul Bert and others, taken with the fact that simple injection of the ear will cause severe vertigo in man, suffice to indicate that any sudden labyrinthine commotion, such as would be induced by an inward displacement of the base of the stapes, or the membrane covering the fenestra rotunda, will initiate the vertiginous state; and thus "experimental vertigo" may be induced by the pressure of a column of air upon the membrana tympani transmitted along the chain of ossicula towards the fenestra ovalis in those who are predisposed to vertiginous affections. It may be regarded as established that the troubles of equilibration in such cases depend directly upon the amplitude of movement of the stapes, or the immobility of the membranous partition of the fenestra rotunda; either condition inducing increased pressure in the semicircular canals. Clinical data fully substantiate this view.

Upon examination of a case of labyrinthine vertigo, if disease of the middle ear be not detected it is usual to infer the existence of a lesion of the labyrinth; yet, to prove that deafness exists apart from lesions of the tympanic cavity is universally acknowledged a difficult diagnostic point, the difficulty consisting in exclusion of affections of the fenestra rotunda and ovalis. Dr. Gellé then discusses the methods of exploration of the labyrinth, and the employment of the diapason,* placed on frontal eminence. He regards the usual assumption that the vibrations are propagated *direct* through the cranial bones to the contents of labyrinth as an error; for he has long shown that sounds thus produced are greatly modified in intensity by altering the state of tension of the transmitting chain of ossicles. Savart and Wollaston have proved the rule to hold good for aerial vibrations. Experiment by centripetal pressure upon the tympanic membrane leaves no doubt upon this point, and decidedly contradicts the views of Bonnenfant, Triquet, Politzer, &c., that diminished auditory perception of cranial vibrations is a good differential sign between enfeebled hearing, and an obstacle to the reception of sonorous waves. It would be more exact to say that this loss of cranial transmission indicates an obstacle to the access of sonorous waves on a level with the fenestra ovalis and rotunda.

He then insists on the value of his test by centripetal pressure, a "diapason" being placed on the frontal eminence of the side to be examined whilst the orifice of the meatus is compressed. In healthy subjects the pressure gradually increased modifies the quality, and eventually extinguishes the sound so transmitted, and a gamut of sensations is thus formed, corresponding to variations in the centripetal pressure, really indicative of the degree of mobility and elasticity of the transmitting chain of ossicles, and especially of the state of the stapes and the fenestræ.

In disease no such modification of sound may be obtained on pressure, or any sound transmitted may be absolutely extinguished upon the *gentlest pressure*, and vertigo and buzzing sounds produced. The conclusion is evident; the transmitting ossicles are immobile and the sound is not modified, or the movement is so slight that the gentlest centripetal pressure renders the stapes immobile and the vibrations are wholly extinguished. The vertigo may be caused by excessive movement of the stapes, or by immobility of the membrane of the fenestra rotunda.

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* By a "diapason" Dr. Gellé would indicate a form of tuning-fork.

The relationship of softening of the membrana tympani to Ménière's vertigo is then discussed; if in such cases the meatus be obstructed, the inward displacement of the tympanic membrane increases gradually, the stapes becomes fixed in a new position, and complete deafness, or at least no modification of sonorous waves, results upon centripetal pressure; *if the meatus be free*, the vertiginous and other morbid symptoms coincide with efforts at swallowing, sneezing and yawning. A case is then recorded where vertigo was induced by very gentle centripetal pressure, and where there was complete deafness both for aerial and cranial vibrations; and yet the membrana tympani and whole chain of ossicles except the stapes had disappeared as the result of suppurative median otitis. In this case the stapes was clearly seen *in situ*, whilst the fenestra rotunda was covered by a cicatricial firm structure. In all cases the labyrinth bridled by the fenestræ is at the mercy of any slight circulatory disturbance. Amongst the many causes of Ménière's vertigo may be cited spasmodic reflex of the tensor tympani under emotional influences due to sudden violent sounds. A typical case of the kind is quoted, in which the vertigo is only occasioned by a noise; such excessive sensibility to noises being explained as due to a fixture of the stapes, and its inward displacement resulting in undue compression of the labyrinthine contents upon the slightest contraction of the accommodative apparatus. Cases are referred to where efforts of attention were always attended by lessened acuity of hearing; here accommodative movements extinguished the transmitted waves.

Heat is another efficient cause of vertigo, some patients suffer only during the heat of summer. Fixture of stapes (as in sclerematous otitis), if accompanied by the least auricular catarrh, may induce compression of the labyrinthine nerves and vertigo.

If on centripetal pressure the transmitted sounds are neither modified, extinguished, nor accompanied by vertigo, we should infer that the stapes is immobile—a condition which may result from inward displacement of the whole transmitting apparatus from old lesions, such as external otitis; yet, the arrest of contripetal pressure may occur at the other end of the chain of ossicles, as in sclerematous states of the membrana tympani, or a bridle may connect one of the ossicles with roof of tympanic cavity; here the sonorous waves are transmitted to the labyrinth, but are no longer modified by pressure, and yet the *stapes is free*.

The second test proposed by Dr. Gellé is what he terms "transauricular auscultation, during centripetal pressure the vibrating diapason being placed on frontal eminence." The ear is examined by the otoscope, and the sonorous waves transmitted across tympanum can be heard by the observer and modified by centripetal pressure. Thus can we distinctly differentiate the distal from the proximal fixture of the chain of ossicles, and so correctly localise the lesion; whilst we check the patient's observations by those of the physician. An analysis of numerous cases of Ménière's disease follows, and as regards the vertiginous state the author concludes that it is an accident and not a necessary accompaniment of the auricular affections, depending on the idiosyncrasy, nervous temperament, extreme reflex excitability, rheumatic or gouty diathesis of the patient. It is especially liable to occur in pharyngeal congestions from obvious reasons. In the large majority of cases of Ménière's vertigo Dr. Gellé finds a grave lesion on a level with the base of stapes and the fenestræ. The author concludes a most elaborate and instructive series of articles by a review of the more important methods of treatment.

In the *Archives of Neurology* for March, Dr. PARINAUD calls attention to ocular paralysis of central origin, and particularly the paralysed condition of the associated ocular movements. Movements of the eye, like other voluntary movements, call numerous muscles into simultaneous action, requiring for this purpose a complete central system of co-ordination which pathological and anatomical facts reveal. Paralytic conjugate deviation of the eyes was described by Foville in 1858, the site of lesion being regarded by him as probably in the pons. Several cases were subsequently reported where this view was confirmed by the discovery of a lesion on a plane with the eminentia teres, implicating the nucleus of the abducens. Then came the researches of Duval, Laborde and Granx, which proved the existence in the cat of a connecting fasciculus betwixt the nuclei of the sixth nerve and that of the third nerve of the opposite side. Lastly, M. Landouzy has further evolved the question by his investigations of the relationships betwixt conjugate deviation and hemispheric lesion. The lateral deviation of the eyes whereby muscles supplied by different nerves are associated in action is probably explained by a similar mechanism to that occurring in associated movements of other parts. Duval has lately announced the existence of a nervous band which unites the nucleus of the sixth and fourth nerves of the same side, and thus the nucleus of the sixth pair gives fasciculi to three motor nerves of the eye. In studying the innervation of associated ocular muscles it is therefore necessary to take into consideration this relationship of individual nerves to multiple centres of origin. The researches of Hensen and Welker indicate that the great nucleus of the third pair extending from the third to the fourth ventricle along the aqueduct, is really constituted of several distinct nuclei having relationships with the different muscles innervated by this nerve; whilst Vulpian showed that the reflex associated pupillary movements (consensual ?) were dependent upon commissural fibres, since they ceased when a very superficial incision was carried along the median line of the floor of the fourth ventricle.

Beyond these connections there are the reflex paths uniting these nuclei with the optic nerves in the nates, according to the researches of Flourens, Ferrier, &c.; the influence of the cortex upon ocular movements has been established by numerous researches and observations; whilst the rôle played by the cerebellum, especially its inferior vermiform process, has been indicated by Duval and Laborde. Such complex relationships demand great caution in the interpretation of facts presented. Our author then proceeds to illustrate paralysis of parallel horizontal and vertical movements; and those of convergence and divergence. His first case is that of a young man, aged 27, who, after many months of severe headache increased by movement, was suddenly seized with sight troubles: in looking to the left, sight is blurred, but he never sees double. A difficulty is experienced in turning the eyes to the left, and there is a tendency to supplement the ocular movement by a deviation of the head instinctively to the left. Paralysis affects external rectus of left eye, and internal rectus of right eye, the former more marked than the latter, and both apparent for monocular and binocular vision. The internal rectus paralysed for parallel movements executes well movements of convergence. The conjugate paralysis is incomplete, there being no apparent deviation to the right when the eyes are in repose, but this becomes apparent after a fatiguing examination. By coloured glass we produce diplopia with all the characters of paralysis of the sixth nerve. Pupils equal, react normally. Visual acuity good; no lesion at fundus. No anomaly of sensibility or motor power in the limbs. Patient is emotional, trembling, and hesitating in speech. Deep reflexes preserved without exaggeration. A few days later vomiting supervenes, with vertigo and violent headache, especially of right side; gait becomes embarrassed, with a tendency to fall to right side. Severe singultus lasting 48 hours, and then laryngeal spasms of some minutes' duration occurring five or six times through night, and threatening a fatal termination. Later on there is loss of power of right side of body. The dynamometer registers, right = 28 kilog., left = 48 kilog. Two months later there is appreciable deviation of eyes to the right, yet convergence is well executed and other movements remain normal. There is right hemiplegia and slight facial paralysis of the *same side*; no alteration of cutaneous sensibility. The instinctive deviation of the head to the left is instructive, and might at first view appear to be an exception to the law of Prevost as to the direction of the head in conjugate deviation of the eyes from lesions in the pons. The unusual implication of the facial on the side of the paralysed limbs, together with the laryngeal spasms, add interest to the above case.

His second observation is that of a lad aged 11, microcephalic, and suffering from chorea and congenital amaurosis, which had been relieved as he grew older. Visual field irregularly contracted; chromatic vision normal; atrophic decoloration of both papillæ. Patient is incapable of turning his eyes to the right, and looking to the left caused nystagmus; all other ocular movements are normal for monocular as well as binocular vision. Nystagmus is only present upon deviation of eyes to left side, and wholly disappears upon median fixation for distant vision and for convergence. Closure of one eye causes marked nystagmus in the other, rendering fixation in any direction impossible.

In the third case there is inability to turn the eyes to the right, the right external rectus and left internal rectus (more especially the former) being paralysed. Convergence is easy but exaggerated for the right eye, on account of the nearly complete paralysis of its abductor muscle. Deviation to left is still more readily accomplished, but is not of normal range; it produces nystagmus, especially of left eye. Elevation and depression of eyeballs normal and unaccompanied by nystagmus; never suffered from diplopia: no deviation of eyes is apparent in repose; no asymmetry of face. Has a tendency to rotate the head to right when he looks in that direction. Pupils equal, and contract normally. No lesion at fundus oculi. Amongst the other symptoms were deviation of the tongue to left and atrophy of its right half.

Dr. Parinaud proceeds to remark, that although paralyses of associated ocular movements are common in disseminated sclerosis, they are usually far less complete than in the cases just described.

The absence of diplopia in these cases often renders the state of ocular paralysis long undetected. The nystagmus, although not peculiar to *Sclérose en plaques*, is almost always associated with the conjugate paralysis dependent upon that pathological condition. Parinaud regards the nystagmus, like that occurring in early infantile amblyopia, as due to a lesion of nuclei in the grey matter superintending associated binocular movements. A difficulty in diagnosis may occur betwixt paralysis of the abducens, and paralysis of the conjugate movement initiated by the sixth nerve. In peripheral paralysis, however, of the outer rectus, the internal rectus of the opposite eye is the seat of exaggerated contraction; so that when the left external rectus is paralysed, if the patient looks to the left side, the healthy right eye deviates suddenly inwards, and fixation is accomplished by the paralysed eye. This differential sign renders the diagnosis simple. Nystagmus is of little import if it takes place in the direction of the paralysed muscles; but when in a direction opposed to these, it becomes a more significant symptom. Lastly, the absence of an alternate paralysis of face and limbs should call our attention to a possible conjugate ocular paralysis, not otherwise revealed by diplopia or persistent deviation.

His fifth case illustrates paralysis of parallel movements in the vertical plane: both elevation and depression of the eyeballs are abolished; lateral movements preserved, but convergence is impossible; the movements of eyelids are intact; right pupil largest; both moderately contracted and reflex contraction to light is abolished. The visual field is normal as regards form, light and colour. Cutaneous sensibility normal; no headache, vomiting or lapse of consciousness had occurred; a tendency to fall to the left side was observed, but is not due to vertigo; slowing of the pulse; urine slightly albuminous; no sugar; loud systolic bruit at base; no atheroma of radials. Recurrent polyuria with slight albuminuria should be considered in relation with the experiments of Claude Bernard, and probably indicate a lesion of the floor of the 4th ventricle, or a reflected irritation from some neighbouring site.

The tendency to fall to the left, with no appreciable paralysis of limbs, also refers us to the Rachidian bulb. As to the associated paralysis of the eye—if it affected but one eye, it might be explained by a lesion partially involving the nucleus of the third nerve, affecting *all the muscles of the globe* innervated by this nerve, but leaving intact the iris and levator palpebræ. According to the researches of Hensen and Welker, this lesion would implicate the posterior part of the nucleus near the upper angle of the fourth ventricle. The absence of paralysis of the internal rectus for lateral movements is readily explained by the fillet which the nucleus of the sixth nerve sends to this muscle.

The sixth observation deals with paralysis of the convergent movements of the eyeballs. An interesting case is quoted of asthenopia of the internal recti: convergence eventually wholly abolished; fixation producing strabismus; alternately covering each eye, the one uncovered executes sharply a movement of fixation; diplopia, once present, has gradually disappeared and can with difficulty be recalled by coloured glasses: *almost complete paralysis of accommodation without mydriasis;* pupils equal, moderately contracted; reaction to light very feeble and no reaction with attempts at convergence. Amblyopia characterised by irregular recession of visual field in both eyes, more pronounced in right eye on nasal side. With + 4 Diopt.: p. p. = 30 Cm.; no dyschromatopsia, no notable ophthalmoscopic lesions. Two months later, double optic neuritis; epileptiform seizures, leaving persistent trembling of arms, lip, and lower jaw. In this case a new growth was found involving the right crura cerebri and cerebelli, implicating the right optic tract, corpora geniculata and right nates and testes; right trochlearis is flattened and softened at its origin; and the right motor oculi is affected in like manner and split into four or five bundles by the pressure of the growth. The lesions were too extensive for an instructive localisation. Donders has shown that in the normal state convergence and accommodative effort is a synergic act. In the above case we see both actions abolished; but we must not conclude that accommodation would be *necessarily abolished* where convergence is impossible: we see the contrary in cases of strabismus, and when it occurs as a result of paralysis of the internal recti; accommodation may still exist, especially if the affection be gradual in its advance or of long standing. Dr. Parinaud concludes a very instructive article by a few further remarks on anomalies of movements of divergence.

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END OF VOL. VI.

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